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OF THE

American Academy of Ophthalmology and Oto-Laryngology

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SEPTEMBER 13-19, 1931

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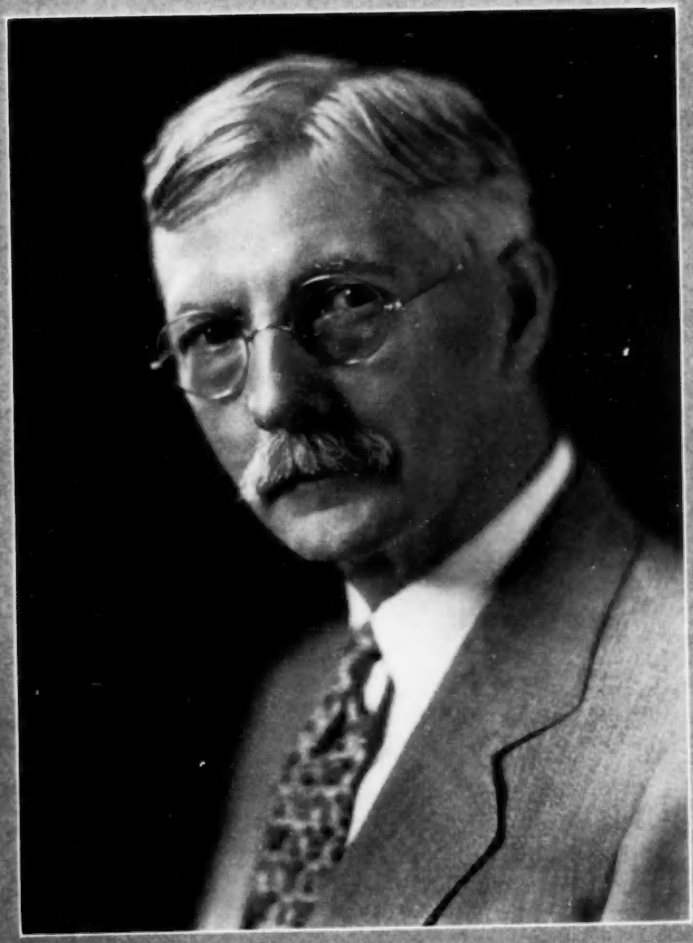

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JOINT SESSION

TABLE OF CONTENTS

PAGE

ADDRESS BY THE PRESIDENT, DR. JOHN F. BARNHILL, Indianapolis, Ind. 15

SYMPOSIUM ON MALIGNANT DISEASES

MALIGNANT DISEASES OF THE NOSE AND ACCESSORY SINUSES 20
DR. JOSEPH C. BECK, Chicago, Ill.

HIGHLY MALIGNANT TUMORS OF THE NASO-PHARYNX AND PHARYNX 39
DR. GORDON B. NEW, Rochester, Minn.

TUMORS OF THE ORBIT 45
DR. BERNARD SAMUELS, New York.

OPHTHALMOLOGIC SESSIONS

TABLE OF CONTENTS

PAGE

Share of Crystalline Lens in Ocular Refraction . 66
Dr. Edward Jackson, Denver, Colo.

Astigmatism—Its Accurate Determination and Correction 75
Dr. C. S. O'Brien, Iowa City, Iowa

An Experimental Study of Detachment of the Retina and Its Surgical Therapy 89
Dr. Leo L. Mayer, Chicago

Angiomatosis Retinæ With Cerebellar Cyst (Lindau's Disease) 106
Dr. Walter S. Atkinson, Watertown, N. Y.

Visual Fields in Chronic Glaucoma—The Effect of Reduced Illumination 115
Dr. S. B. Marlow, Syracuse, N. Y.

Malignant Melanotic Tumors of the Eye: A Study of Histologic Types in 111 Cases 131
Dr. George R. Callender, Washington, D. C.

Cataract in the Eyes of Fresh Water Fishes, Due to the Invasion of the Larvæ of Trematoid Worms . 143
Dr. Park Lewis, Buffalo, N. Y.

The Hyaloid Membrane of the Vitreous . . . 159
Dr. Alfred Cowan and Dr. W. E. Fry, Philadelphia

OTOLARYNGOLOGIC SESSIONS

TABLE OF CONTENTS

PAGE

THE PROGNOSTIC SIGNIFICANCE OF SINUSITIS IN CHILDREN 171
DR. LYMAN RICHARDS, Boston

MANAGEMENT OF THE FRACTURED NOSE 193
DR. E. FULTON RISDON, Toronto

NASAL OBSTRUCTION AND SYSTEMIC CONSEQUENCE . . 199
DR. JOHN J. SHEA, M. A., Memphis, Tenn.

THE DIAGNOSIS AND CONSERVATIVE TREATMENT OF SPHENOID SUPPURATION 209
DR. W. A. WAGNER, New Orleans

SOME EXPERIMENTAL AND CLINICAL OBSERVATIONS ON THE "RETICULO-ENDOTHELIAL" COMPONENTS OF ACCESSORY SINUS MUCOSA 225
DR. RALPH A. FENTON, Portland, Ore., and
DR. OLOF LARSELL, Portland, Ore.

ATROPHIC RHINITIS 239
DR. JOHN A. PRATT, Minneapolis

GENERAL AND OTOLOGIC ASPECTS OF CHOLESTERIN METABOLISM 248
DR. MAXWELL FINEBERG, St. Louis

EARLY INTRINSIC CANCER OF THE LARYNX, DIAGNOSIS AND TREATMENT: OBSERVATIONS ON LARYNGOFISSURE AS A METHOD OF TREATMENT IN A SERIES OF CASES . . 262
DR. GABRIEL TUCKER, Philadelphia

REPORT UPON AN EXPERIMENTAL INVESTIGATION OF THE EQUILIBRIAL MECHANISM OF THE FROG'S LABYRINTH . 282
DR. W. J. MCNALLY, Montreal

TABLE OF CONTENTS

SYMPOSIUM ON AVIATION MEDICINE

PAGE

THE GOVERNMENT ASPECT OF AVIATION 306
DR. L. H. BAUER, Hempstead, N. Y.

PRESENT OPHTHALMOLOGICAL STANDARDS FOR COMMERCIAL AVIATION IN THE UNITED STATES 311
DR. CONRAD BERENS, New York, and
DR. HENRY TEMPLETON SMITH, New York

OTOLARYNGOLOGIC ASPECTS OF COMMERCIAL AVIATION . 351
DR. RALPH A. FENTON, Portland, Ore.

THE PRESENT STATUS OF AVIATION IN THE UNITED STATES TODAY 357
HON. DAVID S. INGALLS,
Assistant Secretary of the Navy for Aeronautics

THE TEACHERS' SECTION

PURPOSE OF TEACHERS' SECTION 359
DR. W. P. WHERRY, Omaha, Neb.

PROBLEMS OF GRADUATE TEACHING 365
DR. DUNCAN MACPHERSON, New York

STANDARDIZING GRADUATE COURSES IN NEW YORK CITY BY THE NEW YORK ACADEMY OF MEDICINE . . . 372
DR. S. J. KOPETZKY, New York City

INSTRUCTIONAL SUBJECTS 377

PRESENTATION OF INSTRUMENTS AND APPLIANCES . . 381

MINUTES, COMMITTEE REPORTS, NECROLOGY, NEW BUSINESS, ELECTION OF NEW MEMBERS . . . 388

ALPHABETICAL LIST OF MEMBERS 427

GEOGRAPHICAL LIST OF MEMBERS 481

INDEX OF AUTHORS AND SUBJECTS 541

JOINT SESSION

PRESIDENT'S ADDRESS.

JOHN F. BARNHILL, M. D.,

INDIANAPOLIS.

I wish to speak briefly of appreciation, gratitude and anticipation.

I appreciate greatly the honor you have conferred upon me by electing me president of this, the greatest association of its kind in the world. I wish also to express my very sincere appreciation of the work of the secretaries during the past year in preparation of the program of this session. One not intimately connected with a labor so far reaching and highly specialized can have no adequate conception of the effort required to provide a balanced collection of scientific papers and an adequate instructional course.

Not only is much work required, but it is work of a kind that can be well done only by those having a wide acquaintance with our members and an experience which leads to anticipation of their wants. I can truthfully repeat now what other presidents have said, that the Academy is fortunate in having the services of such secretaries as Wherry, Benedict, Myers, Gradle and Mullin. May it be able long to keep them.

I wish further to express my appreciation of the committees here in Indiana. Because of their complete, harmonious and effective organization and their unceasing efforts throughout the entire year, the complicated machinery of our local affairs is now ready to function perfectly.

Speaking in another sense, I appreciate very keenly my privilege of association with eminent ophthalmologists and otolaryngologists during my professional career: with those who have passed on; those of mature experience of the present, and those with the enthusiasm of youth who have more recently pledged to the Academy the best that is in them. Looking backward over a period that includes the whole life of this association, I have the feeling which every member no doubt shares, that one cannot attend these sessions without carrying away something that will make him a better doctor and a better man: something of an intan-

gible nature, something akin to inspiration, which enables him to improve his status, to extricate himself from ruts and to work with greater zeal.

Limiting this influence to just a few leaders who have passed from us in recent months, is it not true that each of us owes a debt of gratitude to association with such fine spirits and untiring workers as Ross Hall Skillern, Derrick T. Vail, Clyde C. Lynch and Joseph A. Stucky? Would not each of us be poorer had we never felt the warmth of their hands or listened to their words of wisdom? I have sometimes been asked, "Does it pay to leave one's work and attend meetings of the Academy?" Think what you have received from men like those I have just mentioned, and then each answer the question for himself.

That friendship has a value greater than material things is a universal belief. My last communication from Joe Stucky was filled with this sentiment. To have and to hold it often requires thought and sacrifice. It has been said that friendship is a sentiment more binding even than a king's command. Its cultivation along with the sciences is, therefore, a studied aim of the Academy.

I am sure you share with me a feeling of deep appreciation and gratitude for those far-seeing men who founded this Academy. Some of them are here today where they may behold the giant oak which has grown from the acorn they planted in 1895. Among these founders none is deserving of greater honor than Doctor Hal Foster, who as secretary gave his best efforts to the enterprise until stability was assured.

We appreciate the honors that have come to certain members of the Academy. Three presidents of the American Medical Association have been chosen from our ranks. First, George E. DeSchweinitz, eminent ophthalmologist. Later, Wendell C. Phillips, leader in otolaryngology, and this year the honor has fallen to one of America's best known otolaryngologists, E. H. Carey, of Dallas. These selections prove the exalted position of our specialties and the worth and character of our membership.

In great measure the Academy is a younger members' society. It was intended to be so. To it junior members bring youthful enthusiasm and modern laboratory training in exchange for the clinical wisdom of those more mature in practice. I know of no other important group of specialists in which this arrangement is better balanced or more helpful, and I anticipate an even

stronger tendency in future to give youth its greatest opportunity.

It is not my purpose to discuss recent notable advances in medicine. Merely to mention these would require much time. There has been one recent discovery, however, which merits attention, since it probably touches closely the greatest problems of otolaryngology.

I refer to the experiments of Kendall, who seems to have proved that bacteria really exist in a filterable as well as a visible form. If this be true, it is likely that the filterable form is the one responsible for common colds. It has been long suspected that a filterable virus or an invisible bacteria is the causative agent. Hope is stimulated by the discovery of Kendall that specific measures may soon be available for combating the common cold—undoubtedly the most prevalent and malignant enemy of mankind.

It would be difficult to predict with any degree of certainty what medicine, even in the near future, will be like. Any guess would be a hazard. Everything is moving with such incredible speed, and toward such nebulous ends, that one wonders what the professional problems of our immediate successors may be. We can only believe they will be different, perhaps almost wholly different. It could not be otherwise in an epoch when it is possible for a single factory to make complete more than one automobile at each tick of the watch; when one may see and converse with friends in distant places; when the traveler may breakfast in London, lunch in Berlin, transact business in Paris and return again to London on the same day for dinner. Or when one may circumaviate the globe in an actual flying time of but little more than one hundred hours and when, it is predicted, man in some demon-speed machine may soon race at pleasure with the sun and outdistance it in flight.

As a result of these possibilities, it is not unreasonable to anticipate startling changes in the practice of our specialties. New diseases born in the clouds or miles above the clouds—diseases wholly incident to swift flight and residence in strange and wholly new environments, made available through aviation, will require study and treatment. The Academy, sensing new demands in practice, is wisely devoting a whole period of the present session to aviation problems. This movement undoubtedly is in step with progress.

Another period has been set aside for discussion of the problems of both graduate and undergraduate teaching. This forward-looking movement should interest every teacher and should result in better instruction and a better standard.

A question of importance before all American societies devoted to otolaryngology is that of a library. None of our organizations has any collection of books worthy of mention. It is proposed that all combine to create one central library commensurate with the importance and dignity of our profession. It is the hope that ophthalmologists may join in the movement. I recommend that the Academy appoint one of its members to confer with others already appointed by other societies for the purpose of working out a plan.

I wish to commend the determination of certain universities to establish complete and adequate schools for teaching otolaryngology. It seems probable that Harvard, Pennsylvania, Washington and perhaps other universities soon will surpass all institutions of Europe in facilities and men for teaching these subjects. There is now no more necessity to leave America to study otolaryngology or ophthalmology than there would be to buy steel rails or automobiles. I anticipate the Academy will back and encourage these enterprises with all its power and influence.

The American Boards of Ophthalmology and Otolaryngology have done a notable work. It is not probable that the magnitude of what they have accomplished, and the influence they have had in creating respectable American standards are yet fully realized. It must be clear, however, that the time has already arrived when, to have proper standing and respectability in our specialties, the practitioner must have passed these boards. Holding their certificates gives the public assurance and the doctor confidence. Those not certificated will find they must practice under a handicap for, while there is no law to compel anyone to take an examination, public opinion will soon be stronger than any legal statute.

Medicine today leads other learned professions. While none would wish to make invidious comparisons, or would rejoice at the arrested progress of another, it is human to take pride in our own achievements. While the machinery of justice seems seriously crippled, and the profession of law lags a century behind, medicine, through the untiring energy of its devotees in laboratory and clinic, has climbed to enviable heights.

When the American army overran New Mexico in 1846, fires which had not been allowed to go out in centuries were found burning in captured villages. These were emblematic of a civilization, each generation conceiving it a duty to create a brighter flame.

In a similar sense, this Academy maintains a torch which was lighted by Hal Foster and his little band, and has been kept burning ever since. Those who lighted it and those who have kept it ablaze will soon pass it on to other hands. Faith in the youth of this and future generations gives assurance that the torch will be safe and the flame will be kept burning.

SYMPOSIUM ON MALIGNANT DISEASES.

MALIGNANT DISEASES OF THE NOSE AND NASAL ACCESSORY SINUSES.

JOSEPH C. BECK, M. D.,

CHICAGO.

The Program Committee undoubtedly had a certain object in mind when selecting this symposium of malignant disease in such a small area and distributing it among three essayists, one of whom was to confine himself to malignant disease about the nose and nasal accessory sinuses, the other about the nasopharynx, and the third about the orbit.

This presentation will deal with the neoplasms of the nose and nasal accessory sinuses. It must be borne in mind that it will be somewhat difficult to sharply limit the discussion to this region, as undoubtedly neoplasms having their origin in the nose or nasal accessory sinus may, by their progression invade the orbit or nasopharynx.

It should also be brought out at this time that the discussion will be brief and that details will be, to a more or less degree, omitted, as the time allotted to the dicussion is very short. It might be well to open this subject by presenting and discussing a series of histopathologic specimens of various neoplasms that are commonly observed about the nose and nasal accessory sinuses.*

The first type of neoplasms to be discussed are various so-called precancerous entities. These are, in the main, nonmalignant pathologic conditions, which very frequently form a substratum upon which a malignancy is grafted. One of the very common ones is rhinophyma, a pathologic state that is not in itself malignant but very frequently undergoes malignant degeneration, due to the ingrowth and proliferation of epithelium in the pathologic overgrowth of the glandular tissue of the skin of the nose.

*Forty illustrations were shown on the screen, and an example of one is shown in Fig. 1.

Paraffinoma.—Paraffinoma of the skin about the nose and face, due to the injection of paraffin for cosmetic purposes, is another benign condition that may at times assume malignant characteristics. Most frequently the type of malignancy that follows is of a sarcomatous nature, due to the fact that the irritation is in the connective tissue rather than the epithelium.

Various other benign conditions were exhibited upon the screen showing their histological characteristics, upon which a malignancy might be engrafted.

Cancroid.—One of the most important common dermal lesions of a malignant nature is the cancroid. (Fig. 1.) Microscopic study will show that the basement membrane has been broken through and invaded by epithelial cells. The malignant nature of these cells is well shown by the fact that they vary in size and shape and also in their staining characteristics. One may also note the presence of numerous mitotic figures. Careful examination of the section will show that in the periphery of the lesions there is a great deal of fibrosis, and that this fibrosis, in a manner of speaking, chokes off the lymphatics of the skin and accounts for the very slow spread of cancroid lesions.

The histologic appearance that is exhibited by a cancroid is of practical importance, as it is a matter of common knowledge that this type of lesion responds very favorably to radium treatment. In other words, this is what is known as a radiosensitive growth. It is well known that as many as 90 per cent of the malignancies of this type can be permanently cured by early irradiation alone.

At this time a discussion of the value of the microscopic study of the neoplasm in relation to the diagnosis, course, prognosis and treatment of the disease, is in order. It is only since the recent collaboration of both the pathologist and the radio histologist that one has been able to gain some information regarding the value of the minute picture of the neoplasm as regards the course that the disease pursues and its response to therapy. It has been found that cellular types of neoplasms, which clinically are so highly malignant, and metastasis so readily and extensively are very frequently the most radiosensitive. For instance, it has been found that neoplasms that are embryonal in character, so-called anaplastic lesions, that are highly cellular, and show no tendency towards adult structure, are the ones that are most easily influenced by radio therapy, whether radium or X-ray. And yet, it

is also peculiar that the lesions that show a tendency towards adult characteristics—for instance, the acanthotic lesions of a squamous cell carcinoma, that show pearl formations and regular pavement-like tissue structure—respond very poorly to irradiation. These are best handled by extensive surgical destruction.

Cellular sarcomas, such as small round cell sarcomata and lymphosarcomata, respond very readily to irradiation. On the other hand, fibrosarcomata, carcinomata having a desmoplastic reaction or fibrous infiltration, respond poorly, even to large doses of radium or X-ray. It should also be stated that while the Broder classification of the degree of malignancy is a worth-while study, yet in clinical practice we have been unable to find any correlation between the classification as given by him and the actual clinical manifestations of the disease.

Biopsy.—The question of biopsy is a very important one. Biopsy in the nose, throat and pharynx is comparatively easy, as the neoplasm, as a rule, is accessible. One should remove a sufficient amount of tissue in order to enable the pathologist to make a good preparation so that it can be easily studied. The question of metastasis after biopsy is, of course, a vital one, but we feel, as does Dr. Jaffee of the County Hospital in Chicago, who said, "A biopsy performed with a minimum of trauma, using a cold sharp blade, has very little influence on metastasis." However, in order to safeguard any such occurrence, we have been insistent in telling our patients that should the return of the microscopic section be reported as a malignancy, then we advocate immediate surgical or radiological intervention.

We have come more and more to the point of view held by Dr. New, namely, performing a biopsy on the operating table, getting the report of the frozen section and then proceeding immediately with radiation, or surgery if it is found necessary. The biopsy, however, must be carefully guarded from the moment it is removed from the operating room until it has undergone the technical treatment by the technician. On rare occasions biopsy material has unfortunately been exchanged and a wrong diagnosis returned to the surgeon. Such carelessness is to be deprecated, but unfortunately, as has been stated by Dr. Bloodgood, it has occurred in a few instances.

The gross diagnosis of a malignancy of the nose and nasal accessory sinuses, as a rule, is not at all difficult. The lesion is there, available for inspection, and even palpable with a probe.

However, one must always bear in mind that the clinical differentiation between the various granulomata may at times be not only difficult but also impossible.

We have assumed the dictum that whenever a malignancy is diagnosed or even suspected, a biopsy is always imperative as a confirmatory measure. One may have other signs and symptoms to aid in the establishment of the diagnosis. The X-ray showing peculiar types of encroachment upon the bones of the face, aided by the use of radiopaque oils, may frequently be of help. Such confirmatory evidence as enlarged glands in the neck and the presence of cachexia may also be of assistance. It should be once more reiterated that the diagnosis most frequently rests upon the biopsy and study of the histologic section. The gross pathology of these various conditions is, of course, best studied either on the patient at the time of examination, in the operating room and at times only possible at postmortem.

There is herewith appended a table of malignant disease about the nose and nasal accessory sinuses. (Fig. 2) as observed by us. This is only a partial list of patients actually seen and refer only to those of which we have a compleet clinical record and, as a rule, some photographic evidence—for instance, photographs of patients showing evidence of gross involvement, the progress, as well as the gross specimens, were shown on the screen. An example of such illustration is shown in Figs. 3, 4 and 5.

Many other cases seen in clinical practice at the County Hospital and in consultation, of which no records are available, are, of course, not listed.

Prognosis.—The prognosis of malignant disease about the nose and nasal accessory sinuses is not a very happy one. Even with our modern means of attack, namely, the use of the electrothermic methods of surgery, aided and abetted by the use of radium and X-ray, our percentage of cure leaves a great deal to be wished for. The fact that the anatomic situation unfortunately is in close proximity to vital structures and also that bone is so frequently involved, accounts for this very sorry spectacle that the medical profession has to offer the laity. In contrast to this gloomy picture one should consider that about 90 per cent of the skin epitheliomas, whether basal or squamous cell in nature, respond, as a rule, providing the lesion is seen early, to irradiation.

Treatment.—The treatment of malignancies about the regions under discussion will, of course, vary with the histopathologic nature of the disease. Those highly cellular anaplastic, embryonic types of tumors that tend to metastasis so quickly, as a rule respond poorly to our surgical attempts, and it is in these cases that we may expect something in the future from the use of radium and X-ray. The relatively benign lesions, as shown by histopathology, may at some time respond to surgical intervention.

Surgery.—There have been several types of surgical approach to malignancies about the nose and nasal accessory sinuses advocated. One of the most common and most popular is the Moure operation (Fig. 6) or the Denker operation (Fig. 7). In the latter the exploration and resection is performed under the lip. In lesions that involve the ethmoid and frontal, the modified Lynch operation that permits a radical procedure without too much deformity is advocated, in contradistinction to the Preysing operation (Fig. 8), which however, gives a wider exposure. The oldest, and, of course, most common procedure that has been utilized to attack malignancy of the upper jaw has been the Kocher (Fig. 9) resection of the superior maxilla. With all of these operative measures, it is well to utilize X-ray or radium both as a preoperative and postoperative adjunct.

Surgical Diathermia.—The discussion of surgical treatment of malignancy about the nose and nasal accessory sinuses would, of course, be incomplete without some reference to electrothermic methods. We have been well satisfied by the use of the diathermic cutting current in performing our dissections. In addition to that, we use the coagulating current. In the first place, we employ it to throw a cofferdam of coagulation about the periphery of the growth if it can possibly be performed. We insert the coagulating needle into the uninvaded tissue at some distance from the edge of the advancing lesion and coagulate a regular circumscribing fence about it. Following this the tumor itself may be scooped out by using the cutting loop, or the tumor itself may be coagulated by using the various button or needle electrodes (Fig. 10) and then resort to the use of a curette to remove the débris. By utilizing these few technical procedures the entire visible portion of the growth may be removed. At the termination of the operation or some time after the reaction has subsided we frequently employ a large amount of radium. In the use of electrothermic

methods we would like to refer to a new combined apparatus (Fig. 11) that enables one to employ both the cutting and the coagulating current at one time. There are several other features of this apparatus that are noteworthy in the advance of the technique, and in this I refer to the sterilizable electrodes, the connecting cords, and also to the sterilizable knob that permits the regulation of the current by the operator himself to suit his immediate needs.

Radium.—The use of radium is a very important advance in the treatment of malignancy. A great deal of progress has been made in this, not only in the technic of the use of the radium but also in the study of the relation between the histological structure of a tumor and its response to irradiation. You are all familiar with surface contact and interstitial use of radium (Fig. 12-A, B and C), but I would like to take this opportunity of calling your attention to a new advance in the technic of radium therapy, namely, teleo-radium therapy or teleo-Currie therapy (Fig. 13), as it is called in Europe. In this new technical advance, one utilizes the so-called radium bomb or cannon. (Fig. 13-B.) A large amount of radium, up to four grams, is introduced into a heavy lead container (Fig. 13-B), and the rays from this tremendous amount of radium are permitted to act upon a tumor at some distance from the skin. One may utilize the ray in exactly the same manner as the X-ray therapist, namely, by employing several portals of entry about the skin, all the rays of which are focused upon the tumor lying beneath (Fig. 13-C and 12-D.) Thus one can see that the tumor itself receives a tremendous dose of radium (Fig. 13-E), and the overlying skin only has small individual doses given at the various portals of entry. (Fig. 13-D.) Various technical methods are employed by which the radium is centered exactly upon the growth. (Fig. 13-A), and the best results obtained. If one will spend any time perusing the literature emanating from the Radium-Hemmet in Stockholm and from the Regaud Clinic in Paris, as well as the various radium centers in this country, he will be surprised at the rapid progress being made and the results obtained by the use of radium in the treatment of malignant disease.

X-Ray.—There have also been several technical advances made in the use of X-ray therapy in the treatment of malignant disease. I have specific reference to the use of the new 900,000 volt X-ray tube (Fig. 14), just perfected by Dr. Coolidge, which has been

installed at the Memorial Institute in New York. This apparatus at the present time is in the experimental stage and will be utilized in the treatment of malignant disease at that institution. However, physically speaking, the action of these X-rays under the high voltage tension of 900,000 volts, cannot approach that of gamma irradiation from radium. In order to produce a ray energy that is comparable to the gamma ray emitted by radium one may have to utilize three and one-half million to four and one-half million volts, and as yet these technical difficulties are apparently insurmountable, but probably not for long. The cost to install such a highly complicated apparatus and the large personnel will not, I fear, permit this method of treatment of malignant disease to become very popular. In all probability the tendency will be toward the employment of more and larger doses of radium, although one must be cautious in predicting what the future will hold in store.

The type of deep X-ray therapy that we have employed on the cases we have shown you here today has been of an entirely different form from that mentioned of the extremely powerful machine that is still in the experimental stage. At the beginning of our experience the voltage was not any higher than about 180,000 volts. At the present time the apparatus that we are showing you (Fig. 15) is of a much higher voltage, 300,000 volts, and with it some very good results are recorded. However, thus far we have had no great benefit in using it on malignancies deep below the skin.

In conclusion, it must be emphasized that no one certain definite type of surgical operation, such as the Caldwell-Luc, or the Moure, or the Killian, is ever employed in the classical manner, but must be modified to suit the individual case as it occurs. It should also be stressed that in this region particularly, and with the use of electrothermic methods of destruction of the neoplasm, the question of hemostasis is of greatest importance. For that reason, in an attack on a neoplasm of any considerable size, we have always deemed it a precautionary measure to prophylactically ligate the carotid artery, most frequently the external, but under the stress of obscured anatomy within the neck due to secondary involvement, the common carotid has had to be ligated instead.

There are a number of other factors of importance that must also be stated. This is particularly true in inoperable cases

with extensive metastasis in the neck and into distant parenchymatous organs. In these hopeless cases palliative measures are of prime importance. Pain may not at times be amenable to opiates, and one must, therefore, consider alcohol injections into the trigeminal nerve or even into the Gasserian ganglion, and at times it may be necessary as a measure of last resort to resect the posterior root.

MIXED OR NON-DIFFERENTIATED CELL SARCOMA, HIGHLY PIGMENTED, OF NOSE AND NASAL ACCESSORY SINUSES COMPLICATED BY EXTENSION IN ALL DIRECTIONS, INCLUDING GENERALIZED METASTASIS.

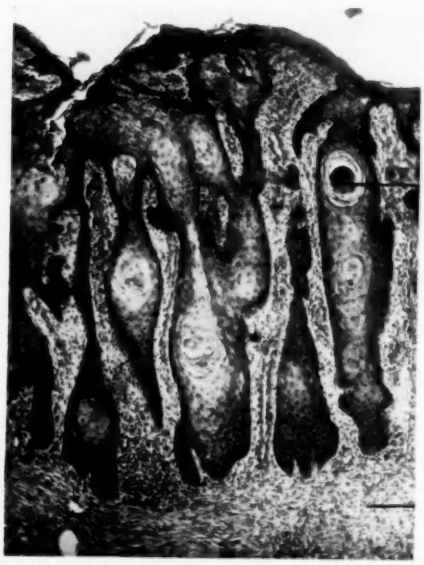


Fig. 1. Cancroid of external nose.

PARTIAL LIST OF NEOPLASMS OF THE NOSE AND NASAL ACCESSORY SINUSES.

	Cases
Rhinophyma	10
Rhinophyma verricosa	2
Nevus angiomatosus	8
Adenoma	2
Cystadenoma	1
Parafinoma	3
Hemangioma	14
Lymphangioma	1
Fibromyxoma (not nasal polypi)	1
Fibrochondroma	2
Osteoma	13
Papilloma	19
Epithelioma	180
Cylindroma	2
Sarcoma	217
Chloroma	1
Plasmacytoma	1
Cholesteatoma	1
Rhinoscleroma	5
Tuberculoma	11
Syphiloma	45
Mucocystoma	8
Teratoma	3
Adamantinoma	1
Dentigerous cyst	21
Meningo and encephalocele	5
Dermoid cyst	2
Endothelioma	5
Total	579

Fig. 2.

Fig. 3. The growth begins to ulcerate near the center of the hard palate. The corresponding swelling over the left side of the face. Radium needles 200 milligrams inserted peripherally of growth, remaining for ten hours. Deep X-ray over left side of face.

Fig. 4. Two weeks later the growth has disappeared with formation of fistula and scars. The swelling on face reduced.

Fig. 5. Approximately one week later. Beginning recurrence with filling in of the fistula.

Fig. 6. Greater progress of growth.

Fig. 7. One week later, still greater progress, blocking the entire left side of the nose. Repeating radium needles and deep X-ray therapy, same dose as Fig. 3.

Fig. 8. Growth receding with greater destruction of hard palate.

Fig. 9. Two weeks later: swelling subsiding.

Fig. 10. Front view, showing still some swelling.

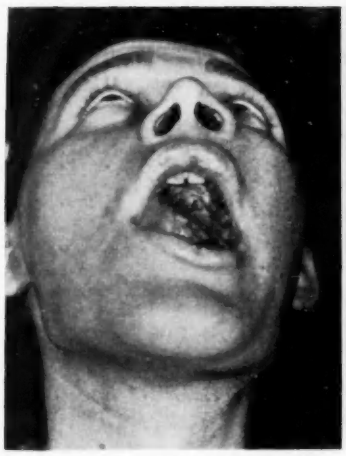


Fig. 11. Approximately one week later, growth showing rapid recurrence.

Fig. 12. Masses removed with partial resection of upper jaw.

Fig. 13. Showing cavity following resection.

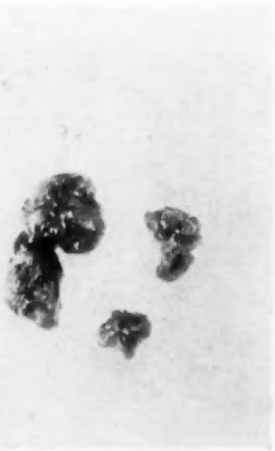


Fig. 14. Masses removed from cavity by radio knife and the remaining cavity thoroughly treated by electrocoagulation.

Fig. 15. Immediate placement of obturator to aid in feeding, speech and prevention of reinfection.

Fig. 16. Approximately one week later, rapid recurrence, showing growth protruding from cavity.

Fig. 17. Same, front view, showing extensive growth on the face. Complete nose block on left side.

Fig. 18. Masses removed at reoperation by curetage and subsequent electrocoagulation.

Fig. 19. Apparent standstill—no further growth. Patient losing rapidly in weight.

Fig. 20. Approximately two months later, recession of side of face. No evidence of growth.

Fig. 21. Acute osteomyelitis and cellulitis of the left side of mouth and face.

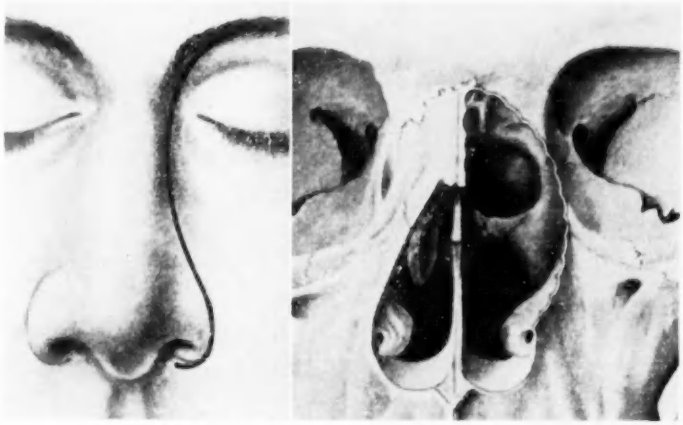
Fig. 22. Approximately two months later. Patient gaining in weight, though greater contraction over the left side of face.

Fig. 23. Approximately four months later. Patient in excellent general condition. No evidence of any recurrence.

Fig. 24. Beginning breaking down of skin covering the side of the face near nose. Small fistula at side of nose.

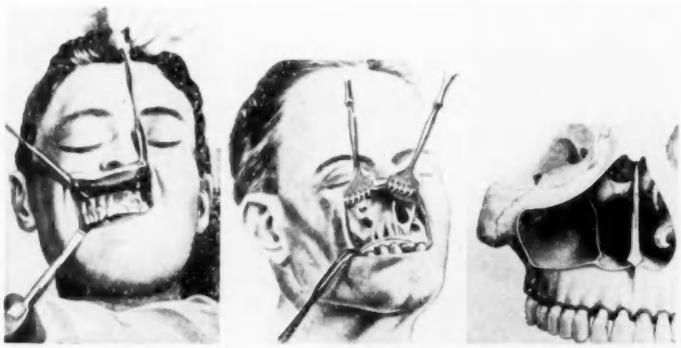
Fig. 25. Approximately two weeks later. Greater loss of substance over the side of face. Marked loss of appetite and weight. Rise in temperature.

Fig. 26. Approximately one week later. Rapid breaking down of the side of the face. General decrepitude.



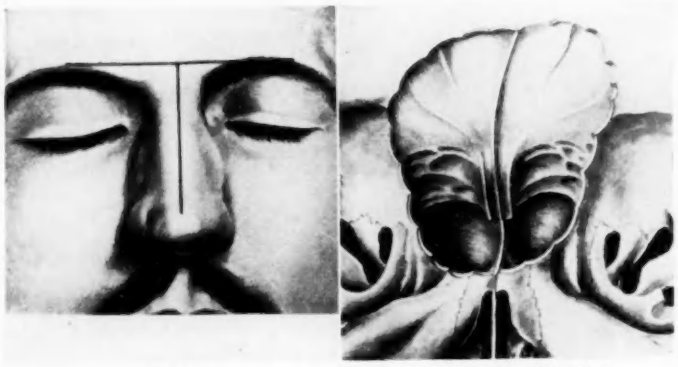
(A) Incision. (B) Complete eviseration.

Fig. 27. Moure's operation.



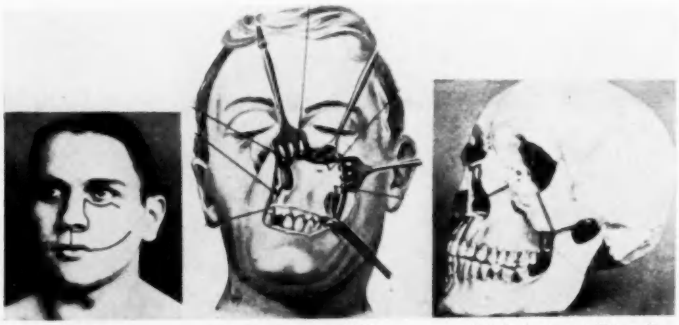
Incision. Superior maxilla exposed. Completed.

Fig. 28. Denker's operation.



Incision. Complete eviseration.

Feg. 29. Preysing Operation.



Incision. Exposure of maxilla. Skull showing line of exsection of the jaw.

Fig. 30. Kocher's operation.

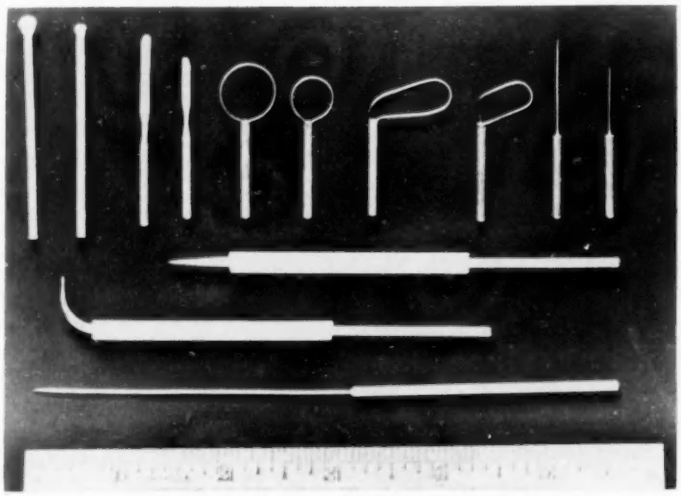


Fig. 31. Various endothermic electrodes.

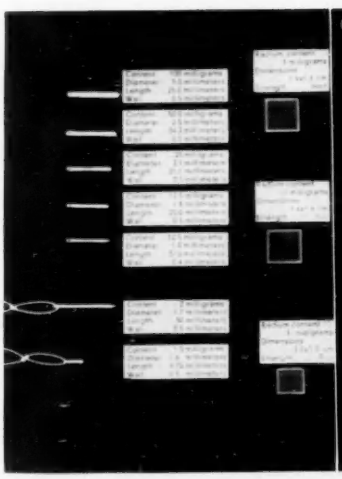


Fig. 32. Radium capsules, Needles, placques and radon seeds.

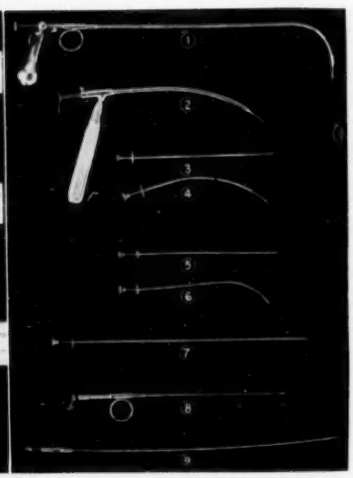


Fig. 33. Radium introducers used for neoplasms about the head and neck.

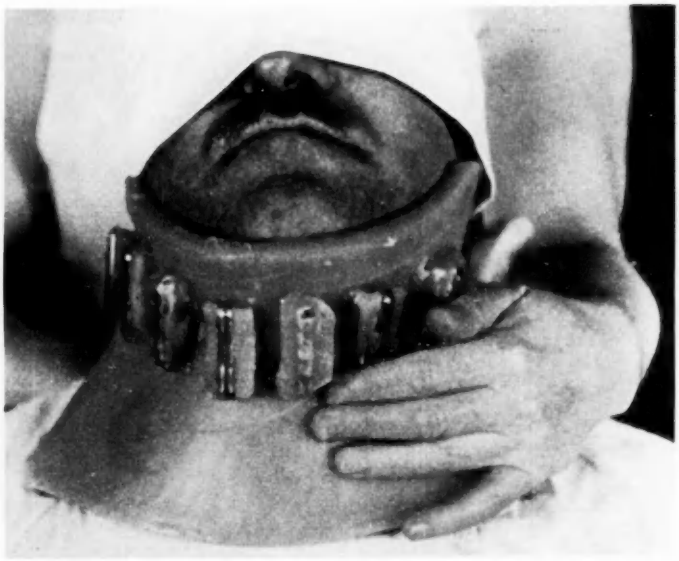


Fig. 34. Radium collar.

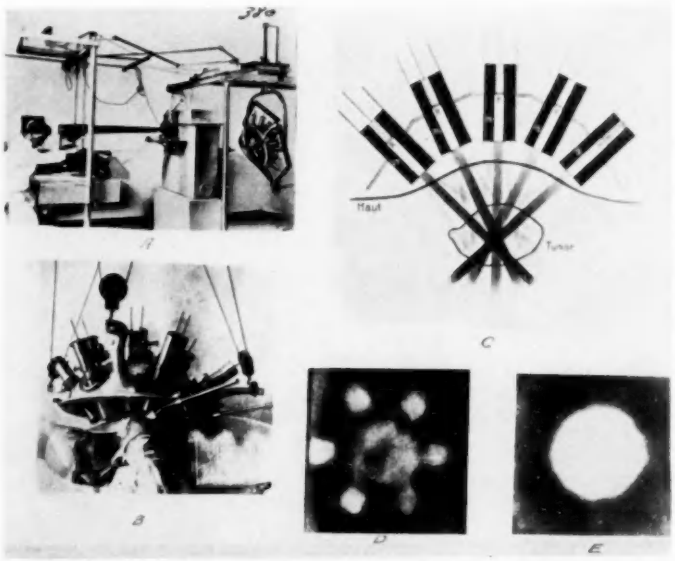


Fig. 35. Teleo-Radium therapy apparatus. Radium bomb.

HIGHLY MALIGNANT TUMORS OF THE NASOPHARYNX AND PHARYNX.*

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ROCHESTER, MINN.

INTRODUCTION.

Malignant tumors of the nasopharynx and pharynx may produce symptoms which cause the patient to consult primarily the ophthalmologist, the neurologist, the internist, the general surgeon or the otolaryngologist, so that unless physicians in general are cognizant of the syndrome many such tumors will not be recognized. The most common malignant tumors in this region are active squamous cell epitheliomas and lymphosarcomas. In a period of fourteen years at The Mayo Clinic 246 patients with highly malignant tumors of the nasopharynx and pharynx were examined. One hundred ninety-one were epitheliomas and fifty-five were lymphosarcomas, of which 194 were nasopharyngeal and fifty-two were pharyngeal.

It has been well known for years that certain of these highly malignant tumors are markedly radiosensitive. In some cases, even in the presence of cervical metastasis, the tumor has disappeared completely under irradiation. Such tumors I have classified as lymphosarcomas and highly malignant epitheliomas, and not lympho-epitheliomas or transitional-cell carcinomas, as has been done by certain European observers. I believe the latter to be in reality squamous-cell epitheliomas graded 4.

SYMPTOMS AND SIGNS.

The symptoms of malignant tumors of the nasopharynx and pharynx are produced by involvement of the structures or of the cranial nerves in this region, so that they may be varied and bizarre, and may be produced by referred pain. About 50 per cent of the nasopharyngeal tumors do not cause the usual symptoms generally referred to as caused by such tumors—that is,

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nasal obstruction or bleeding. The first symptom noted by 42 per cent of the patients was an enlarged cervical lymph node. Epitheliomas usually produce neurologic symptoms or cervical metastasis as the primary symptom, whereas lymphosarcomas, owing to their tendency to grow large, may produce nasal obstruction as an early symptom.

Of the 194 cases of malignant tumors of the nasopharynx and pharynx, 154 were highly malignant epitheliomas and forty were lymphosarcomas. Sixty-one patients (31.4 per cent) had paralysis of one or more cranial nerves. This complication was more commonly associated with epitheliomas in which there were 36.3 per cent involvement of the nervous system than with the lymphosarcomas, in which there were 12.5 per cent involvement.

Although the average duration of symptoms in these cases was fifteen and nine-tenths months, the condition was rarely correctly diagnosed. Usually more than one nerve was involved. In the sixty-one cases 172 nerves were paralyzed (2.81 per cent for each patient). In six cases from five to eight nerves were paralyzed. In three other cases nine nerves were palsied and in one case the frontal lobe had been invaded; this condition is unusual, as the nerves are generally involved after they leave the cranium.

Any of the cranial nerves or the cervical sympathetic nerve may be affected. The nerves infrequently affected are the first, second, seventh and eighth, although disturbances of the ear, such as tinnitus or an obstructive type of deafness, are common as a result of obstruction or invasion of the eustachian tube. The involvement in order of frequency occurs as follows: The sixth, fifth and ninth nerves, the nerves in the jugular foramen, and the tenth and twelfth and sympathetic nerves. In a large percentage of cases the involvement is of the fifth and the sixth nerves and those of the jugular foramen. The sixth nerve was paralyzed in thirty-eight cases. The fifth nerve was paralyzed in thirty cases and the ninth nerve in twenty-four. The cervical sympathetic nerve was involved in four cases, as manifested by Horner's syndrome; therefore, the significant early symptoms of malignant tumors of the nasopharynx are diplopia, facial pain and disturbances in swallowing.

In two cases the cranial nerves were affected bilaterally. In one of these both nerves were paralyzed and in the other the left third, fourth and sixth nerves on the left, and the ninth, tenth,

eleventh and twelfth nerves on the right. Many combinations occurred; when only two nerves were affected the involvement was usually of the fifth, sixth and ninth nerves.

DIAGNOSIS.

The diagnosis of malignant tumors of the nasopharynx and pharynx is usually difficult, and only very careful examination will disclose the condition. A patient recently observed with typical paroxysmal trifacial neuralgia of four months' duration was referred for a radical ganglion operation. This case is illustrative of the difficulties encountered. The patient stated that before the onset of the "quick" pains there was numbness of the lower lip, a symptom suggestive of involvement by tumor rather than of typical trifacial neuralgia, but nasal or nasopharyngeal symptoms were not present. On examination of the nasopharynx a small epithelioma was found, and secondary involvement of the gasserian ganglion produced the typical symptoms of trifacial neuralgia.

The difficulty of recognizing the condition is shown by the fact that 185 operations had been performed for the relief of symptoms of 194 patients with malignant lesions of the nasopharynx previous to their admission to the clinic. The most common single operation was tonsillectomy; this was performed in thirty-nine cases. The largest number of operations was on the nose, of which there were fifty-six, which included operations on the maxillary sinuses and removal of polypi, turbinates, septums and so forth. The mastoid was operated on, myringotomy was done repeatedly, and alcohol was injected for trifacial neuralgia in several cases. Cervical lymph nodes were operated on in thirty-three cases and teeth were extracted in twenty cases.

The symptoms depend on the site of the lesion. Any neurologic involvement, from external rectus palsy to a complete jugular foramen syndrome with involvement of the ninth, tenth, eleventh and twelfth nerves, and the sympathetic nerves, may be the first indication of the growth. The enlarged metastatic lymph nodes are most often confused with tuberculosis, leukemia, syphilis, actinomycosis, cystic tumors of the bronchiogenic cleft, or those originating in the thyroglossal duct, and benign and malignant enlargements of the thyroid gland. Any enlargement of the cervical lymph nodes should suggest a nasopharyngeal examination.

TREATMENT.

In the selection of patients for treatment of highly malignant lesions of the nasopharynx and pharynx, the type of lesion, the site and extent of the primary lesion, the presence of cervical metastasis or intracranial extension are important. The duration of the disease and the amount of previous treatment are also determining factors. It is essential that the patient should be able to return for repeated observation; otherwise, as a rule, little can be accomplished. For this reason, it is much better if they can be taken care of at home or near home.

If selected treatment is carried out there should be a reasonable chance of its benefiting the patient. In observing the effect of palliative treatment on the prolongation of life, it is found that patients who received one or two palliative treatments did not live longer than those who were not treated. The average life of patients receiving palliative treatment was six and nine-tenths months, and those not receiving treatment, five and eight-tenths months; about 28 per cent of the patients were treated. The most favorable lesions were the lymphosarcomas, of which about 40 per cent were treated.

Patients with highly malignant tumors of the nasopharynx were treated by the application of deep roentgen rays externally over the neck, and radium applied directly to the primary lesion. The highly malignant tumors of the pharynx, other than the small lesions which could be removed with surgical diathermy, were treated in a similar manner, sometimes with radium points, needles or seeds directly into the local lesion and deep roentgen rays externally over the neck. In the low grade lesions, which are not considered in this paper, cautery excision or diathermy of the local lesions and removal of cervical glands of the neck give the best results. No doubt some of the poor results obtained in the past in treating such lesions have been due to lack of care in selecting cases and in not grading the lesions microscopically, to aid in determining the treatment. The high grade lesions are more radiosensitive and surgical treatment is of no value in this group.

RESULTS.

Although tumors of the nasopharynx and pharynx are of particular interest from a diagnostic standpoint, the results from treatment present certain points of interest (tabulation). Treatment of these highly malignant tumors usually relieves the patient

greatly and reduces the size of the tumor; sometimes the tumor as well as the glands in the neck disappear completely. Many patients are able to return to their occupation, and with repeated treatments they continue in this manner for several years.

The average length of life of the patients who received treatment (28 per cent) was thirty-four and four-tenths months. One patient lived for ninety-four months and four patients lived an average of seventy-five and five-tenths months. The patients who did not receive treatment lived an average of six and eight-tenths months.

SUMMARY AND CONCLUSIONS.

Tumors of the nasopharynx and pharynx, frequently classified as lympho-epitheliomas and transitional-cell carcinomas, are squamous-cell epithelioma graded 4.

Many unnecessary operations have been done to relieve the symptoms in cases of undiagnosed malignant tumors of the nasopharynx and pharynx.

The finding of a malignant tumor of the nasopharynx in cases of obscure pains about the head and face, frequently clears up the indeterminate diagnosis.

Unless patients with malignant tumors of the nasopharynx and pharynx are seen early, and can obtain adequate treatment, little is accomplished in prolonging life by palliative treatment.

Patients who were treated lived about five times as long as those who were not treated.

TABULATION OF RESULTS

Site	Type of growth	Patients	Total	Patients treated								Patients not treated		
				Traced		Living		Dead					Traced	
				Cases	Av. life, months	Cases	Av. life, months	Good results	Av. life, months	Fair results	Av. life, months	Total	Cases	Av. life, months
Nasopharynx	Lymphosarcoma	40	15	15	34.5	4	75.5	1	54.0	10	16.2	25	21	5.2
	Squamous-cell epithelioma, graded 4	126	31	31	22.2	10	19.7	1	58.0	20	21.7	95	81	7.2
	Squamous-cell epithelioma, graded 3	28	7	7	19.0	2	10.5	..	...	5	23.6	21	19	7.3
Pharynx	Lymphosarcoma	15	6	5	39.0	1	94.0	1	63.0	3	23.0	9	8	6.2
	Squamous-cell epithelioma, graded 4	17	3	3	51.0	1	29.0	2	59.0	..	...	14	12	6.8
	Squamous-cell epithelioma, graded 3	20	4	4	21.0	2	23.0	..	...	2	15.5	16	15	7.3
Total		246	66	65	26.1	20	34.4	5	62.6	40	20.3	180	156	6.8

TUMORS OF THE ORBIT.

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NEW YORK.

Tumors of the orbit are comparatively rare. At the New York Eye and Ear Infirmary, quoting from the annual report for 1930, out of a toal of 35,869 cases treated in the Eye Department, only nine are recorded as orbital tumors. During this period 6,535 operations were performed on the eye, but there were but two exenterations of the orbit. The surgical report of the Royal London Ophthalmic Hospital (Moorfields), for 1930, announces 52,372 outpatients for the year, and yet but one patient was admitted into the hospital with a diagnosis of neoplasm of the orbit. Three exenterations were done during the year. The subject, however, is of prime importance, owing to the gravity of orbital growths from the standpoint of pathology and surgery.

The purpose of this paper is to make a brief pathological review of some of the tumors of the retrobulbar space, the so-called concealed tumors, including those of the optic nerve, all of which sooner or later produce exophthalmos. Attention will be drawn more particularly to the manner in which the pre-existing structures react to the presence of tumors rather than to the morphology of the tumors themselves, concerning which so much has been written. The theme is presented in the form of clinical cases that have yielded anatomical material for study. The tumors are classified according to certain peculiarities as follows:

Group I.—Tumors producing marked inflammatory reaction (of slow growth).

(a) Carcinoma.—No other malignant growth in the orbit is likely to produce so much irritation in the structures invaded. The propagating tumor cells, apparently by the toxicity of their metabolism, are commonly preceded by a lymphocytic infiltration in the adjacent tissues. The result is that bands and membranes of connective tissue, scirrhus, are formed which impede the advance of the tumor. It is striking that the muscles, after the fashion of more highly organized structures, offer little ressitance. In fact, the fibers usually atro-

phy in advance of the carcinoma and in this way they are replaced, so that the function of a muscle may be disturbed out of proportion to the size of the area involved. The propensity of carcinoma to bring about the production of scirrhus has considerable clinical bearing. On account of the contraction of the newly formed connective tissue, the eyeball may be drawn to one side and fixed to the wall of the orbit. This is observed in instances of ulcus rodens of the lid, in old people, when the growth has broken through into the loose tissues of the orbit. In fact, one of the great dangers of surface tumors, on the lids and on the limbus, is their tendency to invade the underlying, more areolar tissues, thus becoming retrobulbar.

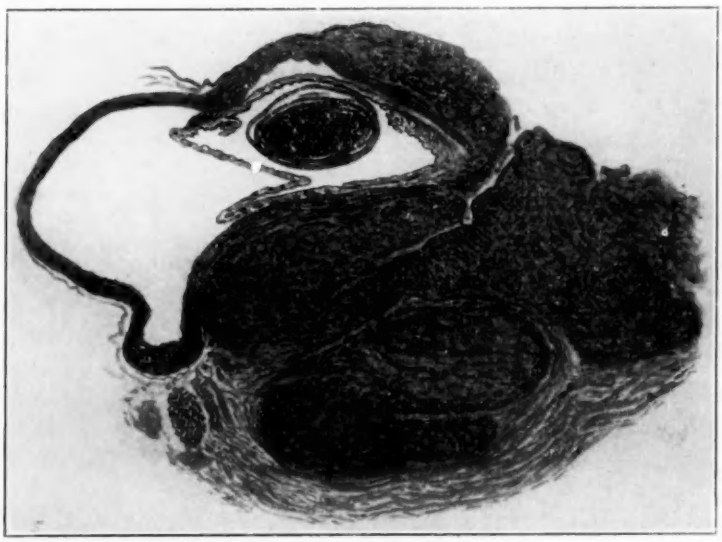


Fig. 1. Epithelioma of the limbus showing inflammatory perforation of the sclera.

An instructive example of malignancy and inflammation going hand in hand is shown in a case of epithelioma of the limbus (Fig. 1), which has extended into the retrobulbar space. Its borders are everywhere outlined by heavy lymphocytic infiltration, and throughout the formation there are bands of newly formed connective tissue. At one point near the ora serrata the inflammation assumed a purulent intensity and destroyed the sclera. Through the opening thus formed tumor cells are about to enter from the orbit into the globe.

(b) Cylindroma.—This tumor is said to originate in the lacrimal gland, or in glandular tissue misplaced in the orbit and not in touch with the gland. Cylindromata belong to the concealed tumors in the strict sense. They resemble basal cell carcinoma not only structurally but in the matter of production of scirrhus. Cylindromata appear not to be so irritating as carcinomata because, according to the preparations, they are not marked by so much lymphocytic infiltration. In both forms the scirrhous ele-

Fig. 2. Cylindroma of the orbit. The scirrhus produced is very pronounced.

ment may predominate. Therefore the exophthalmos resulting from these tumors is likely to progress slowly, and on account of the contraction of the scirrhus it is not apt to attain a very pronounced degree. The indentation of the sclera coincident with the exophthalmos should proceed at a correspondingly gradual rate, being indicated by changes of refraction. Glaucoma is a feature of proptosed eyes of long standing, probably due to interference with the venous outflow. An instance of this is shown

in a case of cylindroma (Fig. 2) in which an iridectomy was performed on a proptosed eye.

Group II.—Tumors producing little or no inflammatory reaction (of rapid growth).

(a) Sarcoma.—Sarcomata are said to be the most frequent of the primary tumors of the orbit. In contrast to carcinomata, they seem to be better tolerated by the tissues normally present which, as a rule, show few signs of inflammation. These tumors commonly lie encapsulated, the thin capsule being often the result of a condensation of the pre-existing fibrous tissue. How rap-

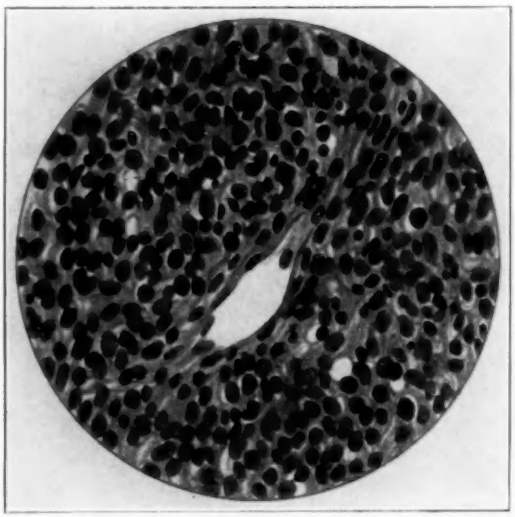


Fig. 3. Retrobulbar sarcoma of rapid development, showing mitotic figures.

idly a sarcoma may enlarge is exemplified in the case of a child of three years. In the short time of fourteen days an enormous exophthalmos developed. There was no sign of inflammation. A biopsy (Fig. 3) divulged a sarcoma. This seemingly disappeared under X-ray and radium treatment. The sharp delineation of a sarcoma is shown in another case (Fig. 4), of sarcoma of the limbus. The growth is making its way backward in an external rectus muscle. The very delicate rim of lymphocytes between the tumor and the muscle in this instance may be the result of irritation from the action of the muscle bundles in a mechanical way. It is to be borne in mind that neoplasms of the

Fig. 4. Sarcoma of the limbus undergoing necrosis in the center. Note sharp delimination between tumors and muscle at the equator.

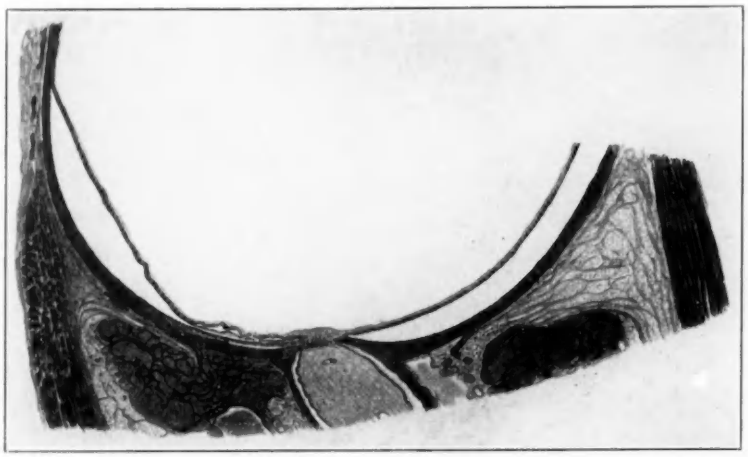
orbit up to a certain stage are in the midst of structures that are frequently in motion owing to the action of the muscles. The extent to which the sclera may be indented by a rapidly growing tumor is emphasized in an example of lymphosarcoma (Fig. 5), in which it is pressed inward on one side from the equator to the optic nerve. Ophthalmoscopically such cases simulate detachment of the retina. The sclera being dense and resistant, does not readily undergo pressure atrophy, but it is not strong enough to prevent changes in its curvature.

(b) Hemangioma. — Vascular tumors are among the rarest ones in the orbit. Occasionally small ones are discovered by accident (A. Fuchs) in microscopical preparations. The cavernous hemangioma shown in this connection (Fig. 6) was found in a girl of twelve years of age. They are dangerous in the orbit because of their tendency to increase in size and to cause pressure atrophy of the contents of the orbit. They may even affect the bony walls of the orbit. The tumor in this case is still confined within the muscular cone. At one point it is in contact with the

Fig. 5. Lymphosarcoma of the orbit producing flattening of the sclera.

dural sheath. There is a slight edema of the papilla. The lymphocytic infiltration in the trabeculæ may be due to irritation from the disturbance of the parts by the action of the muscles. Such irritation, resulting in the formation of connective tissue, impedes the growth of the tumor.

(c) Neurofibroma (von Recklinghausen's Disease). — The preparation (Fig. 7) here referred to is of especial interest because the coiled and enlarged nerves come from the summit of the orbit. In this location, owing to the restricted area, tumors are likely to cause early reduction of vision by pressure on the optic nerve, while at the same time they are pushing the eyeball forward. In this slide one of the nerves has attained a size about twice that of the optic nerve. So far it has increased in thickness, substituting the adipose tissue, but pressure on the optic nerve is imminent.



6. Cavernous hemangioma confined within the muscular funnel.

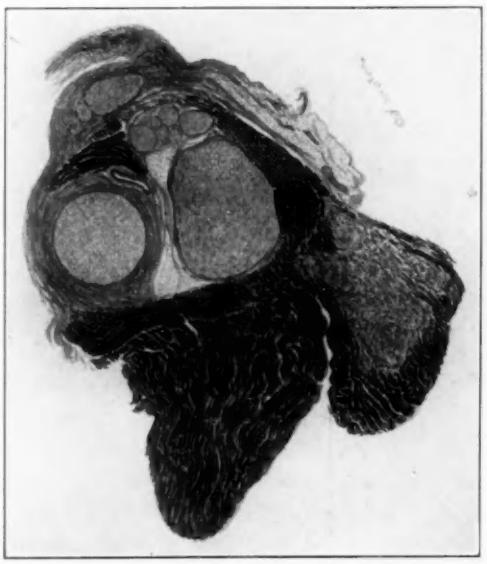


Fig. 7. Neurofibroma (von Recklinghausen's disease) at the apex of the orbit causing straight exophthalmos.

Group III.—Tumors of the optic nerve.

(a) Endothelioma. — A typical endothelioma, still confined within the intervaginal space, is shown in Fig. 8. The space has dilated at the expense of the nerve substance on one side, and of the adipose tissue on the other side. In this example exophthalmos and failing vision probably manifested themselves simultaneously so that the condition would have been difficult to distinguish from an extramural tumor at the apex. The circulation is all the more disturbed because the endothelium has packed

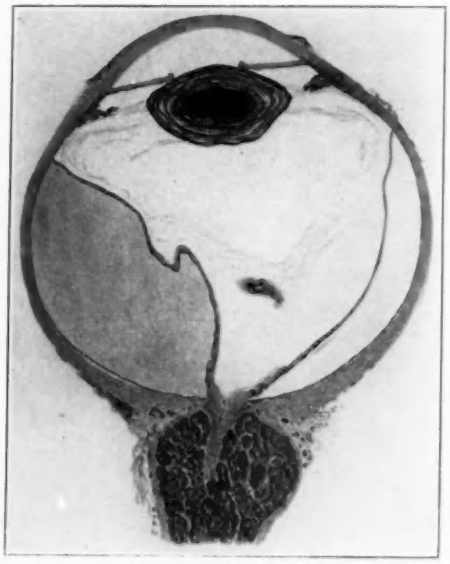


Fig. 8. Endothelioma of the nerve packed into the fornix. Because of disturbed circulation there is edema of the papilla, detachment of the retina and peripheral anterior synechia. The nerve is reduced to a cord surrounded by the tumor. (Courtesy of the Royal London Ophthalmic Hospital (Moorfields).

and dilated the sulcus of the intervaginal space close by the papilla. The clinical changes in the fundus probably came on first with edema and engorgement of the vessels at the nerve head, and at a later stage detachment of the retina developed. The sequence in the ophthalmoscopical picture should have suggested an involvement of the structures in the immediate vicinity of the entrance of the nerve into the sclera, where there are so many blood vessels and anastomoses, causing maximal disturbance of the circulation with exophthalmos.

(b) Glioma.—Although gliomata of the optic nerve originate in the neuralgia, it is known that the vision may be preserved for an indefinite time because of the survival of nerve fibers traversing the gliomatous mass. The growth in Fig. 9 has attained an enormous size, but the structure of the nerve can still be made out as a cord near the center. The exophthalmos must have been excessive, accompanied by pressure atrophy of the orbital contents. The formation is surrounded by a fibrous capsule, but there is no evidence of inflammation. The eyeball has collapsed

Fig. 9. Glioma of the optic nerve producing enormous exophthalmos with collapse of the eye from a perforation of the cornea by an ulcer.

in a characteristic way through the perforation of a corneal ulcer brought on by exposure. It is noteworthy that those who suffer from new growths in the retrobulbar space seldom complain of pain. The explanation may be that there is never great pressure, the globe being pressed forward in a compensating fashion.

Group IV.—Tumors invading the retrobulbar region by extension from within the globe. It used to be thought that all intraocular tumors begin in the orbit and from there invade the eye-

ball. It is now known that the reverse is far more likely to be the case.

(a) Intraocular tumors escaping through the sclera without producing inflammatory reaction. Ordinarily sarcomata attain the retrobulbar space by propagating along the emissaria or by splitting the layers of the sclera. The cleavage of the sclera seems to be caused by some chemical agent which, acting ahead of the tumor, produces clefts, and into the spaces so formed the tumor cells proliferate. The fact that intraocular sarcomata are prone to undergo necrosis plays a great rôle in the method of the extension of these tumors into the orbital tissues. The ne-

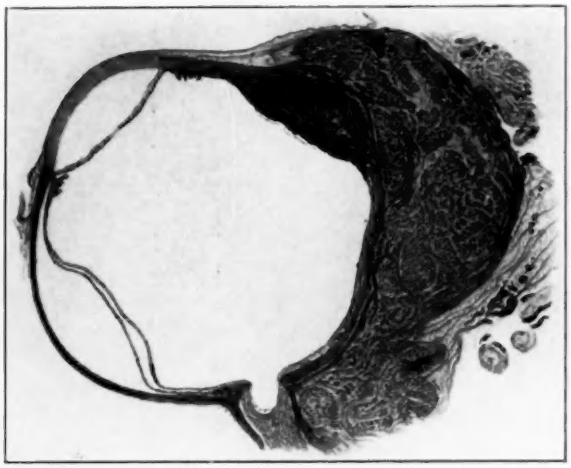


Fig. 10. Infiltrating sarcoma of the choroid surrounding the optic nerve. The extra-ocular nodule is larger than the primary tumor. This growth has reached the retrotarsal space by perforating Tenon's capsule at its posterior extremity.

crotic material being very toxic may set up within the eye an inflammatory process as virulent as that of panophthalmitis, with edema of the lids and exophthalmos. The result may be that an area of the sclera in proximity to the tumor is destroyed by an inflammation of a purulent character, thus forming a place of exit for the proliferation of those tumor cells that may have survived the inflammation. An excellent example of the damage done to the sclera by a necrotic sarcoma is shown in Fig. 10. The small intraocular portion is traversed by bands of connective tissue that represent an organization of the necrosis. The extra-

ocular nodule has dilated the space of Tenon, at the posterior extremity of which it has broken through, freely invading the retrobulbar space. This is an illustration of the destruction of the fibrous tunic of the eye by the action of toxins generated from dead sarcoma cells within the globe, whereas in the case of epithelioma of the limbus in Group I the sclera was destroyed by the action of toxins from living cells on the outside.

Group V.—Metastatic tumors of the orbit.

(a) Carcinoma.—It is well known that metastasis in the orbit may make itself manifest before that in the brain. The seat of

Fig. 11. Metastatic carcinoma of the orbit located in the internal and external recti.

metastatic carcinoma in the orbit is most frequently in the muscular tissue, and generally more than one muscle is invaded, as has happened in the case in Fig. 11, following carcinoma of the breast. The muscles are rich in small blood vessels, and it may be that because of their tortuosity tiny emboli lodge in them. By higher magnification (Fig. 12) the tumor cells are shown to be rather diffusely placed, not compactly arranged as is the familiar way, probably due to the mechanical action of the muscle fibers in activity. There is no inflammatory reaction. A metastatic focus

Fig. 12. The diffuse arrangement of the carcinoma cells is probably due to the mechanical action of the muscle.

Fig. 13. Metastatic carcinoma of the choroid, optic nerve and orbit.

of carcinoma is said not to be so toxic as the primary lesion, which speaks for more rapid growth in the former.

(b) Sarcoma.—Metastatic sarcoma in the orbit is exceedingly rare. In the specimen (Fig. 14) only one muscle is involved in a metastasis from the intestines. It has compressed the muscular structure but shows no tendency to infiltrate it. If visible clinically it may have appeared as a thick cord sharply defined at the site of a muscle on the surface of the sclera without any evidence of inflammation.

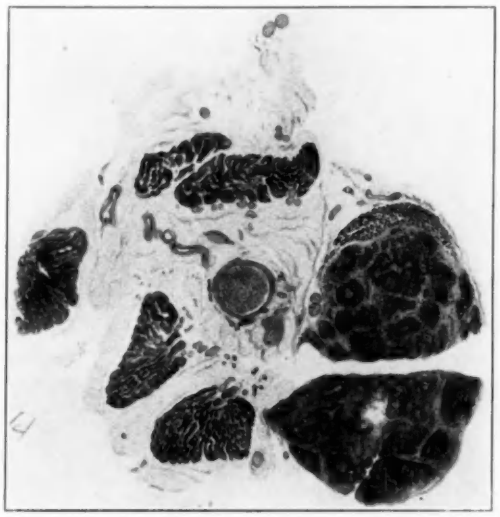


Fig. 14. Metastatic sarcoma in the external rectus from the intestines. Observe the replacement of the muscle by pressure atrophy.

Group VI. Inflammatory Pseudo-tumor of the Orbit.—Clinically, exophthalmos of sudden onset is not an unusual sight. The cause of it can generally be traced to acute infection in the sinuses or lids. However, there is a rarer type of exophthalmos, slow in onset, of a chronic inflammatory origin in the retrobulbar space, that may so simulate the exophthalmos of true tumors that the clinical distinction is well nigh impossible. All told, not a few fatal errors have been reported in the literature in which exenterations were done and the protrusion was found to have been the consequence of a low grade inflammatory process at the apex of the orbit. Benedict collected a number of such cases (Archiv. of Ophth., LII, 1923), and they are constantly crop-

ping up in clinical experience. The following are typical instances of mistaken diagnoses:

1. Male, aged 55 years. Diagnosis: Exophthalmos; papilledema; malignant growth in the orbit. Exenteration. Pathological report (Fig. 15): Papilledema and cystic degeneration of the retina with hemorrhages. The adipose tissue behind the eye is largely replaced by inflammatory connective tissue in places, loose in some areas and dense in others. Here and there are small foci of lymphocytes, some of which have typical germinal

Fig. 15. Chronic inflammation in the retrobulbar space. Note the cystic degeneration and hemorrhages in the retina.

centers. They bear no relationship to the vessels, the walls of which are moderately thickened. The inflammation has permeated through the sheaths of the optic nerve and invades the substance. The entire picture is confined to the retrobulbar space, with the exception of a few scattered lymphocytes along the episclera anterior to the equator.

A blood test, made after operation, divulged a four plus Wassermann. On this account, but not because of any specific pathological features, one is inclined to believe that the inflammation was of luetic origin.

Fig. 16. Chronic inflammation in the retrobulbar tissues with greatly thickened blood vessels.

2. Male. Aged 35 years. Protrusion of left eye three months. Failing vision. Ophthalmoscopic examination: Papilledema and engorged blood vessels at the disc. Wassermann and other tests negative. A biopsy showed inflammation, a fragment of the optic nerve being included in the tissues. Owing to the enormous exophthalmos that ensued after the biopsy, with complete blindness an exenteration was done. Microscopical report (Figs. 16 and 17): Papilledema and cystic degeneration of the retina in the macular region. Indentation of the sclera and participation of its outer layers in a chronic inflammation of the orbital tissues. There is a marked thickening of the walls of the blood vessels. Diagnosis: Chronic, nonspecific inflammation. The microscopic examination of the retrobulbar tissues in these cases of inflammatory pseudo-tumor reveals a peculiar similarity of chronic disease in which there is nothing characteristic by which one could make a definite diagnosis of lues. However, they are on the decrease since the days of the Wassermann reaction and salvarsan.

It is worthy of mention that in the last two cases the pathological report cites cystic degeneration of the retina. Many of the eyes with exophthalmos of whatever cause show on section this

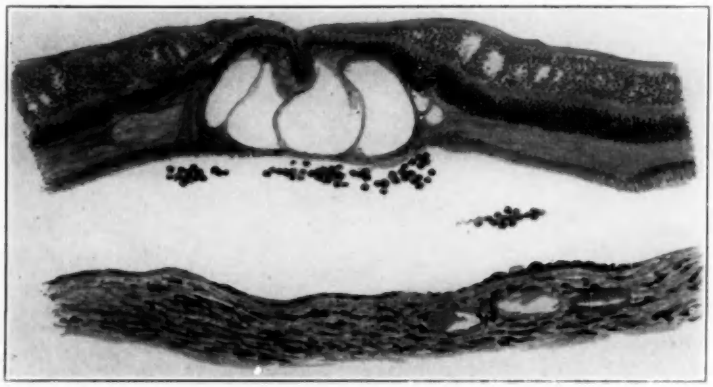


Fig. 17. Cystic degeneration in the macular region of the retina in the above case.

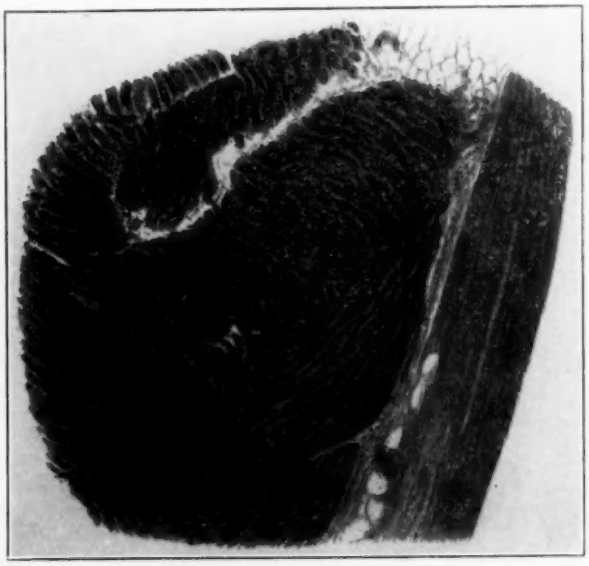


Fig. 18. Focus of lymphocytic infiltration in a case of exophthalmos of unknown origin.

type of degeneration, although it seems to be overlooked and not suspected clinically. It may often be an important factor in the reduction of vision in proptosed eyes.

Group VII.—Enigmatic Cases of Exophthalmos Which Anatomically Divulge no Pathology.—Similar cases are mentioned in the literature (S. S. Golovine, Annales d'Oculistique, June, 1930). In this category belongs this highly instructive case.

Male. Aged 62 years. Exophthalmos for six weeks, gradually increasing to 8 mm. Fundus negative. Normal vision. Wassermann negative (taken twice). After cocainization of the nose, in the examination of the sinuses, the exophthalmos completely disappeared, but it returned and became as pronounced as it was before. On general principles the sinuses were operated upon. Two accidental perforations were made into the roof of the sphenoid, whereupon the sinuses were immediately tamponed. Four days later the patient died of leptomeningitis spreading from the points of perforation. At the postmortem the orbital contents appeared to be perfectly normal. Microscopic examination of the tissues at the apex of the orbit (Fig. 18) shows a focus of lymphocytes among the bundles of the internal rectus muscle and nothing else except the involvement of the sheaths of the optic nerve in the meningitis.

It is possible that Groups 6 and 7 represent those cases of exophthalmos which are known to improve under anti-luetic treatment and even to disappear if given time and opportunity. The surgeon who remembers the innumerable possibilities and pitfalls that may lie at the bottom of an exophthalmos is not so apt to operate rashly as one whose mind is centered only on the malignant aspect of the question.

DISCUSSION.

Dr. W. L. Benedict, Rochester, Minnesota: A group of tumors that has been mentioned only briefly by the previous speakers belongs to the ophthalmological surgical field but have their origin in the paranasal sinuses. In this group are bony tumors of the orbit, usually enostoses, carcinomas, sacromas, hemangiomas, and lymphangiomas. Non-malignant diseases that affect both the orbit and the sinuses, mucocele, gumma, tuberculosis, angioneurotic edema may be present alone or in conjunction with some malignant disease of the orbit or nose and be identified with great difficulty.

I think one of the groups commonly overlooked is the nasopharyngeal mentioned by Dr. New. Paralysis of one of the sixth pair of nerves is often due to an obscure tumor in the nasopharynx. Lowered vision and

field changes are late symptoms of pharyngeal tumors occurring after invasion of the orbit or the region of the sphenoid body. Because of the frequency with which the tumors are overlooked until local symptoms develop they should be carefully sought for in every case of recent paralysis of the external rectus muscles.

Bone tumors are of two types: exostosis which are pyramidal excrescences of bone on the margin of the orbit or at a line of junction of bones of the face; enostoses which grow from the periosteum or subperiosteal bone and are covered with mucous membranes or periosteum, pushing aside all other structures as they increase in size usually as roughly rounded nodular masses of cells, hard septi, spongy material with bone sand and few blood vessels. These tumors are always single, grow from a relatively small pedicle and do not become malignant. They increase only by direct extension usually into the orbit and upward into the cranial cavity. The diagnosis of enostosis must always be confirmed by roentgenograms. The optimum time for surgical intervention is while the tumor is small. Large tumors may be successfully removed but danger of meningitis increases with the extent of invasion of the cranial cavity. The approach for removal of bony tumor is through the orbit. The direction of growth is from below upwards and the extension of the mucous lining affords an avenue for infection so the area is never surgically clean. It is difficult and awkward to attempt to remove bony tumors by way of the nasal passages even after elevation of the nose, while attempt at removal from above through cranial osteoplastic flap as practiced by some neuro-surgeons is followed by an excessive mortality.

Carcinomas of the orbit and nose are more frequent than any other group. The epithelial group, the rodent ulcer, and prickle cell tumor are most frequent and easiest of diagnosis. Many of the pictures exhibited by Dr. Beck in his lecture illustrate this type. Tumors having their origin in the antrums or ethmoids invade the orbit by direct extension and cause exophthalmos, swelling of the lids and often pain about the eye. The origin of tumors in the posterior ethmoid region or the orbit often cannot be determined before operation. It may be presumed, however, that tumors arising within the orbit do not invade the sinuses or if they do it is quite late so that if the evidence of sinus involvement is less than that of orbit involvement, the tumor may more likely rise within the orbit. In either event an orbital approach is to be preferred. I have described elsewhere the method of operating on posterior orbital tumors by an extraperiorbital route, a method which applies equally well to posterior ethmoidal tumor.

Hemangiomas, probably the most interesting group, from the standpoint of origin and diversity of histopathology, stand next in frequency to the carcinomas. Many of them are fibrous, encapsulated masses that do not pulsate and are confined to the orbit. Pulsating hemangiomas usually have arterio-venous connection that permit copious flow of blood through aneurysmal dilatations, cause erosion of bone, congestion of the neighboring soft tissues, and take on malignant characteristics.

Confirmation of the diagnosis of hemangioma before operation is often difficult if pulsation is absent and roentgenogram shows no bone

erosion. If the tumor lies deep within the orbit so that it cannot be felt by the palpating through the lids its location and extent can only be estimated by the amount of proptosis and limitation of ocular movement produced. Treatment by radium and X-rays should precede surgical exploration. Certain types of hemangiomas respond readily to treatment by irradiation, thereby obviating the necessity for surgical intervention.

A rare condition which must be mentioned in any consideration of orbital tumors is pseudo-tumor of the orbit. The soft tissues of the orbit become infiltrated by masses of plasma cells filling the postocular space solidly to the orbital walls with a mass that pushes the globe forward, causes blindness, loss of ocular motility and swelling of the lids. The signs and symptoms are those of malignant tumor of the orbit. The histopathologic picture is that of chronic inflammation, plasma cell infiltration, attenuation of normal orbital fat and muscle and blood vessels. Some pathologists believe the condition is due to syphilis but in a series of cases that I have studied, clinical and serological evidences of syphilis has not been found. Spirochetes never have been demonstrated in the tissue by Warthin's method. Radium and X-ray do not arrest development nor can any effect be noticed after administration of salvarsan, iodides and mercury. I think the etiology of inflammatory pseudo-tumor of the orbit has not been demonstrated. After surgical exploration, even though no tissue is removed the swelling gradually recedes. There is then enophthalmos, with limited movement, and ptosis. The condition has not been known to recur.

Dr. French K. Hansel, St. Louis: This subject has been so thoroughly and comprehensively reviewed that there remains little to be added.

Dr. Beck's discussion and illustrations were extremely interesting and instructive. His extensive experience with these malignant lesions has enabled him to evaluate the various methods of treatment. He has clearly shown that in order to obtain the best results, a certain type of treatment must be instituted in lesions of a certain type and location. He has shown the value of microscopic study of lesions to accurately establish the diagnosis and to determine on that basis the type of treatment to be instituted.

Dr. New's paper clearly illustrates that malignant tumors of the nasopharynx are much more common than generally believed; that in approximately 50 per cent of the cases there are no symptoms referable to the nasopharynx, and that frequently the primary diagnosis is never established. The fact that in 194 cases, 185 operations were performed without discovery of the primary tumor, illustrates the need for a better understanding of this syndrome.

In the diagnosis of orbital tumors, Dr. Samuels and Dr. Benedict have clearly shown that, in view of the rarity of primary tumor of the orbit, the otolaryngologist plays an important part in the determination of the primary site of the lesion. The malignant lesions of the nasopharynx may produce a great variety of ocular symptoms simulating the type of lesion which arises primarily in the orbit.

The diagnostic problem is often presented in which it is necessary to determine if possible whether a tumor arises primarily in the orbit, or

extends to this location from the sinuses, the nasopharynx, or through the sphenoidal fissure from the middle fossa.

I want to take this opportunity to compliment the authors for their extremely interesting papers which have been presented in a most interesting and comprehensive manner.

DR. EDWARD JACKSON, Denver: Perhaps the best tribute that can be paid to those who participated in this symposium is that the only criticism that can be thought of is that they presented too much valuable new material for any audience to master in one session. It is wonderful, however, to be brought in contact with so much that is new in our daily work. We need to be reminded of that often.

I was fortunate enough to see the cases Dr. Benedict referred to of inflammatory pseudo-tumors of the orbit, and very puzzling they were—puzzling when the sections were under the microscope, and certainly very puzzling at the first presentation of the cases. That kind of material is only appreciated by studying and following it from year to year. As a starting point for future work on carcinoma, this morning's program is extremely valuable.

DR. JOHN F. BARNHILL, Indianapolis: This discussion should not end without stating what I believe is a fact, namely, that malignancy in its earliest stages is a local disease. If attacked by sound surgical methods at that time, it is about as curable as other surgical affections. At least, that seems to be the experience of surgeons who operate early.

It cannot be emphasized too often that early diagnosis and early operation are wholly necessary to good results in the treatment of malignancies.

DR. JOSEPH C. BECK, closing: I think the greatest compliment we have had is from Dr. Jackson, namely, that there was so much presented that men cannot begin to discuss. I think we will have a continuation of the discussion around the dinner table. That is usually the way—men hesitate to get up and say what their experiences are. If I sense anything at all in the way of what we should like to discuss, it is the modern treatment of malignancies. It is not the treatment by radium that I wished to show in those pictures, not the radium bomb, nor the treatment by X-ray, but the great thing is early diagnosis. If every man would examine the postnasal space with his mirror, many of these growths that Drs. New and Hansel mentioned would not be missed. It is not always easy to do—patients do not like it—it takes time. I believe the Michel's mirror has been forgotten by many laryngologists but students should learn to use it routinely as they do a nasal speculum, and if they do they will find these cases. Dr. Hansel recently presented a series of cases showing the value in early diagnosis by the aid of postnasal examination and of doing a thorough biopsy, thus knowing what you are doing. Then you may put your hope in the future of this wonderful substance, radium. There is no standard price of radium, but the way Belgium is producing the material I think with better times later on we will have plenty of radium to treat cases with.

I have been interested to hear of the osteomata, of the extension from the sinuses, and being treated through the orbit. Of course, the meningi-

tis which is so much feared is the thing that makes it act like malignant growth, but if you allow some of the osteoma to remain more will follow.

I have enjoyed this symposium very much.

DR. BERNARD SAMUELS, closing: I wish to take this opportunity to thank those who made such favorable remarks in regard to our part of the symposium.

I agree with Dr. Benedict that not so many of these pseudo-tumors are observed today as in years ago. I think we owe a debt of gratitude to Dr. Benedict for much of our knowledge of pseudo-tumors, because he was one of the pioneers in this work.

OPHTHALMOLOGIC SESSIONS

SHARE OF CRYSTALLINE LENS IN OCULAR REFRACTION.

EDWARD JACKSON, M. D.,

DENVER, COLO.

Many advances of science are quickly applied to medicine and surgery, but this has not been true of optics. That the crystalline lens was important, in the refraction of the eye, has been known for 300 years; but how large a share it takes in ocular refraction has remained unrecognized. Since Thomas Young,[1] 100 years ago, showed that the lens changed in shape with accommodation, there have been numerous articles to support different theories of accommodation; but very little light has been thrown on the share of the lens in the static refraction of the eye.

Removal of the lens for high myopia was proposed by A. Weber,[2] in 1858. In 1887, Fukala[3] did such an operation, and in 1889 he reported sixteen cases. Ten years later such operations were discussed by Cross[4] and others, before the Ophthalmological Society of the United Kingdom. The numbers reported by that time ran into hundreds. Any case carefully measured before and after operation would have added to our knowledge of refraction of the crystalline lens. But those who are engaged in working miracles by operations have little time or interest for exact scientific measurements. By 1900, the amount of myopia to be corrected in a certain case by removal of the lens[5] was estimated by various methods at anywhere from 12 to 29 D. More knowledge of lens refraction is to be expected, from the careful measurements of eyes for glasses, following up the changes that occur when patients return.

To understand the influence of the lens on the refraction of the eye and its changes, we must keep in mind the development of the crystalline lens.[6] By the time the embryo has reached a length of 3 mm., the thickening of ectoderm constituting the "lens plate" is clearly visible. At the end of the first month, the lens vesicle

has formed and has separated from the surface ectoderm; and the cells of its posterior wall have begun to elongate, to develop the lens fibers. At the third month the suspensory ligament, through which the shape of the lens is to be controlled, has begun to form. Collins[7] found that at the fourth month the transverse diameters of the lens and the eyeball were one-half what they were at birth, and one-third of those diameters in the adult. The whole eyeball has reached its full size at about five years. But like other parts derived from the epiblast (the epidermis, the nails, hair and brain), the crystalline lens continues to grow throughout life. Priestley Smith[8] found that in the period from 25 to 65 years the lens "adds one-third to its weight, one-third to its volume and one-tenth to its diameters."

Other changes occur in the lens as it grows older. Its shape changes from near spherical toward a flattened disc. Its consistence, at first a soft, almost uniform jelly, becomes firmer, until the older part, the fetal nucleus, becomes solid, sometimes almost as resistant as horn. Its elasticity, supposed to reside in the lens fibers, that in early life could be held flattened by stress of capsule and suspensory ligament, and take their natural curve when released, is lost. Instead there remains a rigid nucleus, and a soft, unelastic, even mushy cortex. The index of refraction, or density to light, is at first uniform throughout the lens, but becomes higher in the nucleus, while remaining low in the later developed cortex, until in the end each layer of the lens has an index different from that of the deeper and more superficial layers that adjoin it; and the problem of the theoretic refraction of the lens becomes too difficult for even the mathematical experts.

The study by Collins of the development of the accommodative power of the eye, through the suspensory ligament of the lens with its intercrossing fibers, is supported by the later investigations of Miss Ida Mann.[6] The assembly of all these interacting factors, that play their part in the physiology of the crystalline lens, contrasts strongly with the simple hypothesis of Helmholtz, of an elastic lens, flattened by the compression of its capsule, under the stress of the suspensory ligament. Even the recognition that not the lens but its capsule possesses the required elasticity, which is dominated by the greater thickness of some parts of the capsule than others, that has been worked out by Fincham,[9] only adds to the complexity of the subject. The struggle between flattened shape and greater refractive index, between increased size of lens

and the greater influence of its small nucleus, between cortex ever being added and cortex disintegrating, as it does in later life, must arrest attention, and bring out the importance of the influence of the lens on refractive changes in the eye.

The knowledge of lens refraction, to be gained from the usual methods of clinical study, is obtained by comparing the total refraction of the eye, measured by skiascopy and subjective testing, with the corneal refraction, which can be closely estimated with the ophthalmometer. Despite the confusion arising from the discussion of obliquity of the lens and the greater curvature of the posterior surface of the cornea, Gullstrand[10] thinks the difference between total and corneal refraction is to be set down as lens refraction. The effect of the obliquity of the lens is as much a part of the lens refraction as is the increased effect of a convex lens held obliquely before a screen for experiment. The insignificant influence of the shorter curve of the posterior surface of the cornea is equally a part of the corneal refraction. Their existence and possible influence are matters of academic interest which have no practical bearing on the refraction of the eye as we study and correct it.

The ophthalmometer of Javal and Schiötz differed from the laboratory instruments that had preceded it, in that it was capable of general use in the clinical measurement of refraction. It has now been in such use for more than forty years. Anyone who has cared to do so has been able to accumulate records of the corneal refraction. Few such records have been published; almost none since the first decade of clinical ophthalmometry. These records of corneal refraction cannot be as accurate as those of the total refraction of the eye, as measured by skiascopy and subjective tests. With such records we must observe standards of practical accuracy and usefulness.

Gullstrand,[11] who is well qualified to speak of mathematical accuracy, says: "It almost goes without saying that the errors of measurement with modern ophthalmometers, even in the case of long practice and highest skill on the part of the observer, are not to be lightly estimated. The collimation error, indeed, with the instruments now in use, ought not to be more than one-fourth diopter and may be rather less when the figures to be collimated are advantageously constructed. But the focusing error, which depends in great measure on the skill of the observer and the

repose of the patient, will not be certainly below this value except under the most favorable circumstances, so that as a usual thing the possible error may be estimated at one-fourth and one-half diopter. While these errors are comparatively unimportant for purposes of ordinary practice, they cannot be left out of account in the more precise measurements of corneal astigmatism or in the measurements of the radius at several points in one and the same principal section." It may be noted that the apparent "spherical" refraction of the cornea is affected by the focusing error of the ophthalmometer, and may easily vary a whole diopter, even when taken by the same observer, using the same instrument.

In this paper standards previously chosen[12] have been used: "Changes in amount, all of 0.50 D. or over, changes in the direction of the principal meridians of more than 10 degrees, where the astigmatism amounted to 0.50 D. or over." These are close to the possible limits of exactness set by Gullstrand, and have been based on clinical experience in the use of improved ophthalmometers. Many records of ophthalmometric observations give only the amount and meridians of astigmatism. Those of the writer have always included the spherical refraction of the cornea as indicated by the ophthalmometer. As given here this does not mean the curvature of a single meridian; but the mean, or average, of the two principal meridians.

A comparison of cases that have been measured by the ophthalmometer, at from three to five different times, showed that no closer accuracy than the above was possible. Even records of the same eye measured twice in the same month, or on successive days, will show variations up to the above limits. A typical case record, of a man whose refraction has been followed from the age of 27 to 55 years, is instructive. Each record was obtained without reference to previous records and with the same instrument.

OPHTHALMOMETRIC READINGS.

Case I:

	Right Eye			Left Eye		
1902	45.00	0.50	95°	44.75	1.00	85°
1908	43.75	0.75	90°	43.75	0.75	90°
1914	44.25	1.00	90°	44.50	0.50	70°
1920	44.50	1.00	90°	44.50	0.50	45°
1930	45.00	0.50	100°	45.00	0.50	80°

The same variability of readings, but essential constancy of corneal refraction, is shown in a girl and woman under observation from 10 to 49 years of age:

Case II:

	Right Eye			Left Eye		
1890	41.50	1.12	85°	42.00	1.12	90°
1908	40.00	2.00	90°	40.00	1.75	90°
1930	42.25	2.00	90°	42.25	2.00	90°

A woman observed from 64 to 78 years of age showed:

Case III:

	Right Eye			Left Eye		
1903	45.50	0.50	90°	46.00	0.50	90°
1913	45.00	0.25	75°	46.00	0.25	85°
1917	45.50	0.50	10°	46.00	No cylinder	

The corneal refraction is here of importance for estimating the refraction of the crystalline lens. It rarely changes as the patient grows older. In 644 eyes the spherical refraction, as taken with the ophthalmometer, has not changed more than one diopter in any of the 626 eyes. It had changed only in 22 eyes.

Of these eyes the corneal refraction had increased in 12 and diminished in 10 eyes. The average age of patients in whom it had increased was 32 years; and in those in whom it had diminished, 41 years. A general survey of other cases seems to show a slightly greater tendency to increased corneal refraction in early life.

Changes of one diopter or more, in the amount of corneal astigmatism occurred in 44 eyes. The greater changes were in young people. Only three were in the eyes of patients over fifty, and the greatest change in these was 1.50 D. In younger patients under 25 years there were four eyes that changed in corneal refraction as much as 2. D. to 3.50 D., the average being 2.75 D.

It has been shown before[12] that the great mass of changes of the total refraction of the eyes occur after the age of fifty. The 76 cases in which three to five observations were made with the ophthalmometer showed no certain or material change in the corneal refraction. All required, in the same period, from two to ten changes in their correcting glasses for distance. The average time they were observed was 17.37 years. The average number of changes required was 4.24. The average time between changes was a little over four years.

Of the changes that occurred in the glasses required by these 644 eyes, in 449 eyes were changes of one diopter or less in amount. This means that in many cases a change in astigmatism was due to increased refraction in one principal meridian, and diminished refraction in the other, the amount of change not being over 1 D. in either. In only thirteen eyes was there no curvature change of over 0.25 D. in either meridian; but in seven of these there was a notable change in the direction of the principal meridians. In six eyes there was no change of astigmatism.

The number of eyes in which the change of refraction was from 1.25 D. to 2. D., inclusive, was 149. Eyes showing a change of 2. to 3. D. numbered 18; those that changed from 3 to 4 D. numbered 5; between 4 and 5 D., there were 3; between 5 and 6 D., only one case, and one other case of between 7 and 8 D. In the whole series of 644 eyes there were only nine eyes that had not changed at all in their refraction in 8 to 18 years, an average period of eleven years. In five cases one eye had changed, but the other remained the same. In only two patients had there been no change in either eye.

In so far as our methods of measurement allow us to judge, in all of these cases except the 22 in which some change of the cornea was found, the change in refraction was due to change in the crystalline lens. Very slight variations in the form of refractive index of the lens are capable of causing the moderate, or low, errors of refraction, that produce nerve strain through the eyes, that has been popularly called eyestrain. This series, taken in connection with those reported on by Dr. O'Rourke and myself[12] to the American Ophthalmological Society, show that changes in refraction occur frequently at all periods of life. By the exclusion of other causes for such changes, it is established that the great bulk of them are due to changes in the crystalline lens.

When a patient returns ten times in twenty-seven years to have his glasses changed, and each time the change gives relief from his symptoms, the only rational conclusion is that the symptoms depended on the change in refraction. One such case is more conclusive than a hundred that would not wear glasses, or would not wear them constantly, or wore glasses a little while that did not accurately correct the refractive error. Many such cases can be overlooked by the doctor who doubts the good of glasses or who is loose and inexact in his methods of measuring refraction.

Cases like those studied in this series exist. They are common enough to constitute a large and important part of the work of the ophthalmologist. They show the practical importance of the crystalline lens in the refraction of the eye. The changes in refraction they represent should be studied by measurement of the refraction, and such studies should be associated with examinations of biomicroscopy, the progress of partial cataracts or their regression, when this occurs. From several points of view the changes in the crystalline lens are important, most so in connection with the changes in refraction that they produce.

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DISCUSSION.

Dr. Alfred Cowan, Philadelphia: The eye of the infant is so short that if it had nearly the refractive power of the adult eye it would be axially hyperopic 18 or 20 diopters. Since the eyes of infants are generally emmetropic, or nearly so, there must exist, therefore, what might be called a compensating curvature myopia of 18 or 20 diopters. Dr. Jackson's findings are in agreement with other investigators in that this excess of refractive power of the eye of an infant must exist in the crystalline lens, since the radius of curvature of the cornea of an infant hardly varies from that of an adult. As the eyeball grows in size with the rest of the body of a growing child, the dioptric system normally decreases in power. The great number of emmetropic individuals proves how nicely the balance between the dioptric power of the system and the length of the eye is generally maintained. When we think that such a small variation as 1 mm. in the length of the eye will make a difference of nearly 3 diopters in the refraction, the success in so many instances of

this tendency of the eye to maintain emmetropia throughout life is undoubtedly one of the greatest wonders of nature. If the rate of growth of the eyeball is too slow for the decrease in refractive power, we have hyperopia; and if the reverse condition exists we have myopia. Simple myopia is a refractive anomaly due to a too rapid growth of the eyeball. That it takes place during the school age is to be expected. This is the growing age. I think it has nothing to do with class work.

The curvature of the cornea, the depth of the anterior chamber, the indices of refraction of the cornea, the aqueous, and the vitreous, are all very exactly known. Less is known of the index of refraction of the crystalline lens. In the first place, the index is not the same throughout; the index cannot be measured directly on the living eye and there is no doubt that there is a certain postmortem change.

It is impossible to ascertain the refracting power of the lens except in the living eye; but direct measurements cannot be made with any degree of accuracy and the only method that is left is to calculate it from the loss of refraction that takes place in the eye when the lens is removed. Generally the average aphakic eye requires a correction glass of about 10 or 11 diopters. On this basis the lens systems of all schematic eyes are estimated; but up to the time of Gullstrand no investigator considered correctly the connection between the change of shape of the lens and the amount of accommodation.

Gullstrand, by his exhaustive work, has shown us the character of the refraction of the lens. Since the medium of the crystalline lens is not homogeneous, he studied the optical imagery in heterogeneous media. He found that, instead of a continuously varying index of refraction of the lens substance, the lens consisted of a series of iso-indicial surfaces of discontinuity, the index of refraction of which increases from the surfaces to the center; that during accommodation the lens not only changes its shape, but actually increases its total refractive index.

The dioptric power of the lens system of most schematic eyes is too high. In Gullstrand's schematic eye the power of the lens, reduced for air, is about 19.1 diopters, but by its position it actually adds only about 15.6 diopters to the total refractive power of the eye in air. So that, reducing this power for the index of refraction of the vitreous, the principal point refraction of Gullstrand's aphakic eye is about 12.6 diopters of curvature hyperopia. If the second principal point of the correcting glass is placed 14.5 mm. in front of the cornea, the required power would be about 10.7 diopters.

This does not mean, however, that if the eye were myopic to such a degree that a —10.7 diopter lens in the same position would correct it, that the removal of the crystalline would render it emmetropic. It should not be forgotten that the correcting lens placed before an ametropic eye cannot change the dioptric power of the eye. The lens merely changes the direction of the incident light so that it comes to a focus at the fovea.

Basing the calculation on the constants of Gullstrand's schematic eye, an axial myopia which is corrected by a —10.7 diopter lens at a distance of 14.5 mm. in front of the cornea will have its punctum remotum at a

distance of 109.3 mm. in front of the first principal point of the eye—a principal point myopia of about 9.15 diopters—and this eye will be about 27.5 mm. in length. After the removal of the crystalline it would be corrected by a +5 diopter lens placed 14.5 mm. in front of the cornea.

By the same method of calculation and with the same constants, an axial myopia which would be corrected by a —18 diopter lens in the same position would have a length along its axis of about 31.5 mm. and the posterior principal focal distance of Gullstrand's schematic aphakic eye is 31.031 mm. This eye, therefore, would be almost emmetropic after extraction of the crystalline, and would be corrected by a —0.7 diopter lens.

These results are almost exactly in agreement with the clinical findings.

Dr. Edward Jackson, closing: I have nothing to add. The paper was intended to open the subject, and Dr. Cowan's remarks in discussion opened it wider than was possible within the limits of the paper.

It is a subject that has practical bearings, important enough to warrant the individual who is practicing ophthalmology in becoming interested in it; and giving considerable time to the study of his own records, that are much more convincing than reading any book, or the studies of someone else. Study your own records of cases. That is the only way to convince yourself of some of these things.

ASTIGMATISM—ITS ACCURATE DETERMINATION AND CORRECTION.*

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The accurate determination and correction of astigmatism is of utmost importance for every oculist. There are comparatively few cases of hyperopia or myopia uncomplicated by astigmatism, and it is usually just this latter element which gives rise to most of the patient's symptoms and which is most difficult to correct. Many patients are comfortable with glasses which properly neutralize the astigmatism, even though an associated hyperopia may be undercorrected or a myopia overcorrected. But if the astigmatic correction is inaccurate the discomfort persists.

Regular astigmatism is produced by an unequal radius of curvature in the different meridians of the cornea, and a similar condition in the crystalline lens. In such astigmatism, rays of light from a point source do not focus in a point on the retina; the rays of light passing through each meridian of the eye come to an approximate focus (points focus in a line) either in front of or behind the retina at different points along the optic axis. In an astigmatic eye there are two principal meridians, one of which has the strongest refraction and the other the weakest. Rays of light passing through the strongest refracting meridian have the shortest approximate focus, while those passing through the weakest refracting meridian have the longest approximate focus. Between the focus† of each of these two principal meridians lie innumerable foci from other meridians situated between them. The point at which the retina bisects this anteroposterior line of foci determines the type of astigmatism—i. e., simple hyperopic, compound hyperopic, mixed, simple myopic or compound myopic.

In an astigmatic eye the retinal image is always blurred, since the approximate focus of only one meridian is on the retina, even

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†It is to be understood that the term "focus" indicates only an approximate focus. In astigmatism the image of a point is, at best, a line.

under the best possible conditions. This blurring is due to the fact that the foci of all meridians except one are either in front of or behind the retina, consequently, instead of a sharp focus, the image is formed of diffusion ovals, circles or lines.

Of the two principal meridians the stronger refracting one is usually vertical or within 45 degrees of the vertical, while the meridian at right angles has the weaker refraction and is horizontal or within 45 degrees of the horizontal. This being the most frequent type of astigmatism, it is known as astigmatism with the rule. While this type of error is more frequent in young persons and adults, such is not the case in senility, since here the stronger refracting principal meridian often lies nearer the horizontal position. This is known as astigmatism against the rule.

In an astigmatic eye the accommodation is constantly changing, shifting from one to the other principal focus, especially in eyes with low myopic or low to moderate amounts of hyperopic astigmatism, since the patient attempts to correct the poor vision by varying accommodative effort. In high errors this is not true to such an extent because the patient realizes that, no matter what amount of accommodation is exercised, vision remains poor. This constant shifting of accommodation in an effort to recognize the form of objects occurs most frequently in astigmatic errors of two diopters or less. The amount of astigmatism may change with the shifting of the focus for distant and near objects as a result of unequal contraction of the ciliary muscle.

Few asthenopic symptoms occur in myopic astigmatism, except in low errors, for accommodation is not brought into play to such an extent as in hyperopic errors. The patient realizes that the exercise of accommodation makes the vision worse, consequently the ciliary muscle is allowed to remain in a state of relaxation. These patients squint the lids together and in this manner cut down the size of the diffusion lines or ovals, thus improving their vision to a certain extent.

In hyperopic astigmatism of high degree poor vision is present, but ordinarily reflex symptoms are not pronounced because the correction of the error requires too great an effort of accommodation and the patient is not willing to exert himself constantly to such an extent. In low and moderate degrees of hyperopic astigmatism many symptoms are present as a result of the constant

activity of accommodation. The ciliary muscle is never quiet, and an unquiet accommodation gives rise to many symptoms. The eye focuses between the extremes of the two principal meridians, and there is constant variation back and forth from one to the other in an attempt to recognize objects at which the shifting gaze is directed. In astigmatism the patient sees better when vertical lines are focused on the retina. The vertical lines are more important, especially in the recognition of letters. This necessitates maximum accommodation in the hyperopic individual, since vertical lines are focused by the horizontal or weaker refracting meridian. After the vertical lines are focused the lids are drawn almost together, and in this manner diffusion lines are shortened, thus improving the vision. Since the patient with hyperopic astigmatism must use maximum accommodative effort to see most distinctly, and since the accommodation is unquiet, many symptoms of eyestrain result. There is also an element of fatigue resulting from the effort to recognize and interpret the constantly changing images. Unequal contraction of the ciliary muscle in its different segments may also be responsible for many symptoms. In addition to the usual complaints, such as headache and ocular distress, persons with astigmatism frequently complain of diplopia, apparent movement of objects, dancing objects, vertigo and referred pains radiating into the shoulders and neck.

DETERMINATION OF ASTIGMATISM.

One can often recognize the presence of astigmatism after having heard the complaints of the patient.

The vision of an astigmatic eye is peculiar in that, whereas the hyperopic or myopic person reads to a certain point on the visual chart and stops almost immediately, the patient with astigmatism confuses and miscalls letters in several lines, seemingly regardless of their size. Certain confusion letters, such as X K H N M W, A R B P F T E or O D Q C G U are miscalled in several lines. This peculiar feature of vision is due to inability to see distinctly and simultaneously lines of a letter running in different directions; certain parts of a letter are seen distinctly and a guess is hazarded at the others. This results in confusion of similar letters.

With the ophthalmoscope moderate and high degrees of astigmatism are easily recognized, and the amount of the error may be roughly measured. The vessels of the fundus are not all dis-

tinct when viewed with the same lens. In the higher grades of astigmatic error the nervehead may appear oval. Starting with a high plus lens the area of the nervehead and macula is observed and, as the strength of the lenses in the ophthalmoscope is gradually reduced, the retinal vessels running in one direction become distinct. A note is made of this lens and the vessels at right angles are next brought into focus. The difference in the strength of the lenses with which the two sets of vessels are observed is an approximate measure of the amount of astigmatism. One must not forget that the lens with which the vessels are made clear is a measure of the refraction at right angles to these vessels. Consequently the vessels really indicate the axis of the cylinder to be used in correcting the astigmatism—e. g., if the vessels running vertically are seen best with a + 4 diopter lens and the vessels running horizontally are seen best with a + 2 diopter lens, the astigmatic correction is a + 2 diopter cylinder, axis vertical.

While the ophthalmometer does not enable us to measure accurately the total astigmatism of an eye, it is useful in many instances. With this instrument one can measure only the curvature of the anterior corneal surface, and even this is not measured over the pupillary area but slightly to either side of it. The greatest curvature of the cornea is usually in the vertical meridian, while the greatest curvature of the lens is more frequently in the horizontal meridian. Thus the lens usually counteracts to some extent the corneal astigmatism; consequently in low and moderate errors of corneal astigmatism with the rule, the total astigmatism is less than that indicated by the ophthalmometer. In high corneal astigmatism with the rule the total astigmatism is usually higher than that found with the ophthalmometer, since in these eyes the upper and lower portions of the sclera lie closer to the lens than is normally the case, thus relaxing the suspensory ligament above and below and increasing the vertical curvature of the lens. In corneal astigmatism against the rule, the total error is usually greater than the ophthalmometer reading. Likewise, the axis of astigmatism, as measured on the ophthalmometer, is not usually absolutely accurate; only the axis of astigmatism of the anterior corneal surface is indicated, and when the lenticular astigmatism is taken into account the axis is often slightly different than that registered on the ophthalmometer.

In the average case dynamic skiascopy gives a close approximation of the astigmatic error. This is especially true if an

attempt is made to relax the accommodation by making the test in a dark room and having the patient direct the gaze at a distant point of light.

Manifest refraction is often unsatisfactory, especially in patients with an active accommodation. Due to the unquiet accommodation and to an unequal contraction of the ciliary muscle an improper estimation of the astigmatism and its axis is not infrequent. In myopes the usual dynamic method is followed, while in hyperopes the fogging method is probably best. One must be careful to prevent squinting or head-tilting, since both are employed by the patient as a means of improving vision. In the fogging method the patient is made artificially myopic by the addition of a spherical lens which is of greater strength than that lens which corrects the weakest refracting meridian, and the strength of this lens is gradually reduced until one set of lines on an astigmatic dial appears distinct. Then a weak minus cylinder is added with its axis parallel to the indistinct lines and the cylinder is gradually increased in strength until the lines of the entire dial appear equally distinct.

A cycloplegic is indicated in all patients except elderly persons with little accommodation. One should be sure that the accommodation is well relaxed during the skiascopic and subjective tests. As a routine cycloplegic scopolamin in 1/5 per cent aqueous solution serves well. It produces excellent relaxation of the ciliary muscle and is of much shorter duration than atropin. Homatropin is effective in some eyes but often proves unsatisfactory.

Skiascopy is carried out by means of spheres and cylinders.* This method of skiascopy with cylinders is an accurate method for the determination of astigmatism. In an eye with simple hyperopic or myopic astigmatism only cylinders are used. In an eye with compound hyperopic or compound myopic astigmatism the meridian requiring the weaker lens is neutralized with a sphere, and in this way the remaining error is converted into a simple astigmatism. Now all that remains is to neutralize the astigmatism of the eye with a cylinder of the same strength and axis but of opposite sign. In an eye with simple astigmatism or

*For a more complete description see Retinoscopy with Cylinders, American Journal of Ophthalmology, February, 1926, Vol. 9, No. 2.

after neutralization of the first meridian with a spherical lens in an eye with compound astigmatism, a band of light remains which moves with the mirror in hyperopic astigmatism or against it in myopic astigmatism. The long axis of this band indicates the axis at which the correcting cylinder is to be placed. In the case of hyperopic astigmatism a plus cylinder is placed in the trial frame with its axis parallel with the long axis of the band of light. If myopic astigmatism is present a minus cylinder is used with its axis parallel with the long axis of the band of light. Remembering that only the astigmatic error remains and that a cylinder is now in position which may or may not correct this astigmatic error, one of the following conditions is present:

Cylinder of correct strength and at the proper axis—neutralization in all meridians is present.

Cylinder at the proper axis but too weak—the band of light moves in the same direction as before and remains at the same axis, but the light is more brilliant and the movement more rapid. Correct by increasing the strength of the cylinder until the point of neutralization is reached.

Cylinder at the proper axis but too strong—in this case the band remains in the same axis but the direction of movement is reversed. Correct by decreasing the strength of the cylinder until the ocular astigmatism is neutralized.

Cylinder slightly off axis but of the proper strength—here the band of light, although retaining its same direction of movement, shifts to a position about 45 degrees to the opposite side of the proper axis. Also a new movement of opposite direction is created in the meridian at right angles to the above band of light. Correct by rotating the cylinder a few degrees toward the new axis of the original band of light.

Cylinder slightly off axis and too weak—the same conditions hold as in the previous case, with the exception that the changed axis of the original band of light lies less than 45 degrees away from the proper axis. Correct by increasing the strength of the cylinder and rotating it a few degrees toward the new axis of the band of light.

Cylinder slightly off axis but too strong—here again conditions are the same as in the above two instances except the axis of the original band is over 45 degrees away from the proper axis. Cor-

rect by decreasing the strength of the cylinder and rotating it a few degrees toward the new axis of the band of light.

The subjective test under cycloplegia is used as a final check and may or may not be as accurate as the skiascopy with cylinders. In certain patients the subjective method is of no great benefit; this depends somewhat upon the reaction of the patient and somewhat upon the eye. In certain astigmatic eyes which have remained uncorrected for many years there may be an amblyopia for certain lines, usually the horizontal ones, and the patient may be unable to co-operate because of poor visual acuity. The correction which has been found with the retinoscope is placed in the trial frame and the patient is asked to read the chart. If the visual acuity is good, we know that the findings are correct or nearly so. In myopic astigmatism minus spheres and minus cylinders are used throughout. The axis is carefully checked, then the sphere and cylinder are each reduced in strength by about one-half diopter; following this the necessary additions are made by alternately holding before this partial correction a minus .25 D. sphere and a minus .25 D. cylinder and choosing that lens with which better vision is obtained. Finally, the axis is rechecked under reduced illumination. In hyperopic astigmatism, after the visual test with the combination sphere and cylinder as found by the retinoscope has been completed, a transposition is made and a plus sphere and minus cylinder are placed in the trial frame and the axis is checked. Then the original plus sphere and plus cylinder are reduced in strength by about one-half diopter and by the alternate use of a plus .25 D. sphere and a plus .25 D. cylinder the correction is strengthened until the greatest acuity of vision is obtained. Finally, the axis is checked under reduced illumination. Jackson's crossed-cylinders may be employed instead of the above method.

After the influence of the cycloplegic has worn away, a post-cycloplegic test is made, but here no change is made in the cylinder. Even though the patient believes the cylinder better at a different axis, it is a wiser policy to retain the same cylinder axis as found under a cycloplegic.

TREATMENT OF ASTIGMATISM.

If the patient is to be given relief the astigmatism of the eye must be properly neutralized—i. e., the strength and axis of the correcting cylinder must be accurate. It is often wise to place the

combined trial-case lenses in a lensometer in order to determine exactly the strength which the combination will have when ground into one lens.

If one must err in the correction of astigmatism, it is much more comfortable and better for the patient if the cylinder is slightly undercorrected. In this case, if the cylinder is off axis, either as a result of poor refraction, ill fitting glasses or head and eye movements, the axis of the newly created astigmatism will lie near the old one; while if the cylinder is too strong and off axis, the axis of the new astigmatism lies over 45 degrees away and causes an entirely new and unaccustomed burden to be thrown upon the ciliary muscle.

When the glasses are worn the head and eye movements must be taken into consideration. If the head is turned upon an antero-posterior axis the eyes compensate for this by turning in the opposite direction and, if lenses are being worn which correct the astigmatism properly while the head and eyes are in the normal primary position, the rotation of the eyes behind the immovable glasses produces an entirely new astigmatism with its axis in a different position. In case the correcting cylinder is slightly under-corrected the new axis lies nearer the proper axis than in the case of an overcorrection and consequently does not cause the patient so much trouble.

If the correcting cylinder is too weak but at the proper axis the patient will probably receive more or less relief. If the cylinder is at the proper axis but too strong, the relief will not be as complete, but neither is as bad as a cylinder with its axis in an improper position. In the latter instance, not only is there created a new astigmatism at a new axis but the spherical correction is also affected. The axis of this new astigmatism, created by an improperly placed cylinder, is 45 degrees or less away from the proper axis in case the cylinder is of proper strength or too weak. But in case the cylinder is too strong the axis of the new astigmatism lies more than 45 degrees away and trouble may be expected. The above phenomena are due to the fact that two cylinders, one in the eye and the other in the glass, are rotated on each other and create a new spherocylindrical combination, with the axis of the new astigmatism coinciding neither with that of the eye nor with that of the glass but situated at some distance from them.

In children the full cylinder, no matter how high, is always prescribed. The vision usually improves after a correcting glass is worn. The eye may be amblyopic for certain lines and vision improves when the images of these lines are clarified.

In an astigmatic error of one diopter or less always give the full correction but do not overcorrect. Patients ordinarily have no trouble in wearing such a correction, since the new astigmatism created by head and eye movements usually amounts to less than one-eighth diopter, and the axis of the new astigmatism thus created is less than 45 degrees away from the proper axis if the cylinder in the lens is correct or slightly too weak. In high astigmatism the full cylinder may be prescribed if the patient has worn correcting glasses previously, but in case not even a partial correction has been worn before, the cylinder is reduced slightly. The head and eye movements, in persons with a high error which is fully corrected, create a new cylinder of considerable strength and distant axis; this condition may cause discomfort. But if the cylinder is slightly undercorrected the new axis created by head and eye movements lies closer to the original axis and the lens may be worn with more comfort.

Cylinders with oblique axes are quite difficult to wear; this is especially true in adults who have not worn glasses previously or in those who have been improperly corrected. Prismatic action results from the placing of cylinders with oblique axes before the eyes. In order to do away with this prismatic action the correcting lenses are placed very close to the patient's eyes, the optical centers of the lenses are lowered 3 or 4 mm. below the geometric centers, and the pupillary distance of the lenses between these lowered optical centers corresponds exactly to the patient's pupillary distance. The visual line in both distant and near vision usually falls below the horizontal, and by lowering the optical centers of the glasses to a position through which the visual axes usually pass, prismatic action is avoided. The pupillary distance of the lenses is placed between these two lowered optical centers. In a few rare cases two pairs of glasses are necessary if strong oblique cylinders are prescribed—one pair for distant vision and another pair for close work, each of which has an accurate pupillary distance corresponding to the pupillary distance of the patient in distant and near vision.

In spherocylindrical combinations having a low sphere and a high cylinder it is usually necessary to reduce the sphere more

than in combinations in which low cylinders exist. Some patients may see better with crossed cylinders.

Glasses for the correction of astigmatism are placed as close as possible to the patient's eye; the lenses are set pantascopic in order that the line of vision may pass at right angles through the lenses. The lenses should not be round, for they are liable to turn in the frame and change axes; if a round lens is prescribed a diamond scratch is placed at the temporal side and the patient is instructed to keep this mark in its proper position. Frames must fit well and have sufficient strength, since bent frames change the axis of a glass which in the beginning properly corrected the astigmatic error. Nose glasses should not be allowed. In other words, every precaution is taken to keep the cylinders in proper position before the eyes.

If care is exercised in the determination and correction of astigmatic eyes the patient may be given relief from many unpleasant symptoms.

DISCUSSION.

Dr. C. P. Clark, Indianapolis: A dependable technic for the measurement of ametropia is of the utmost importance to all ophthalmologists. The measurement of ametropia does not give to the surgeon such a thrill as he receives from various surgical maneuvers, or an experience of spectacular accomplishment such as he enjoys at the operating table; but the accurate determination and correction of errors of refraction exact more of a conscientious surgeon's time than is demanded by any other service which he is called upon to perform. The satisfaction of patients will reach the same levels of appreciation for the correction of troublesome errors of refraction as will be expressed for the success of many of the common ocular operations. People wish to see clearly and comfortably. It is necessary that the ophthalmologist be painstaking in his examination, and that he employ those methods which yield the best results. Assemblies of physicians should frequently review methods of refraction and emphasize those procedures which are of more recent development. The subject which Dr. O'Brien has presented to this assembly is a most important one and of everyday interest to all of us.

In order to determine the proper lens prescription for a patient, consideration must be given to the following subjects: (1) the use of cycloplegic drug; (2) the objective determination of the amount of ametropia; (3) subjective methods of refraction; and (4) the determination and adjustment of the binocular muscle balance.

It has been my experience that the use of efficient cycloplegic drugs in all patients under the age of 50 years enables me to make a closer estimation of the refractive state of the eye. The old rule that patients over 40 years of age can be satisfactorily refracted without cycloplegia is

not a good rule because there are many patients past this age who have too much ciliary muscle activity to be fully refracted without suspending the accommodation. Difficulty is experienced in determining the amount and axis of astigmatism. It is a common experience to find patients who have been examined without cycloplegic and whose astigmatism has not been properly determined. The examiner should endeavor to determine the near point of accommodation for all patients, for he will occasionally uncover an unexpected degree of ciliary muscle activity in patients who are past 50 years of age. A minimum amount of effective cycloplegic drug should be employed in the examination of these patients. A miotic drug should be instilled into the eye when the examination has been completed to guard against any possible elevation of intraocular tension.

The instruments for objectively determining ametropia are the ophthalmometer and the retinoscope. I do not feel that the ophthalmometer is of great value except in aphakia. The retinoscope is of great value when used by a skilled examiner. There are many retinoscopes, simple and complicated; the simplest are the best.

At the objective examination the lenses used are spheres or cylinders, or both. The strength and axis of astigmatism can be more accurately determined with combination of spheres and cylinders than with spheres alone.

At the subjective examination I have not found the astigmatic dial charts to be of much value. Letters and numbers which are easily confused are my best test objects, and especially so when they are illuminated with light of low intensity. The use of crossed cylinders, as advocated by Dr. Jackson, is an invaluable aid. Should the patient become confused or fatigued, the examination should be interrupted.

An examination for glasses is not well done unless consideration is given to the ocular muscle condition, but this phase of the subject is not under consideration.

The portion of the ametropia which is of the most importance, in my opinion, is the existence of the astigmatism, and it is unusual to find a patient who does not have an astigmatic error.

Dr. Louis Bothman, Chicago: I have recently submitted to the Archives of Ophthalmology a paper on the comparison of homatropine and atropine as cycloplegics, in which I was able to corroborate some of the statements made by Dr. O'Brien today. I shall present at this time a brief resume of the findings on astigmatism.

Dr. O'Brien said in his opening paragraph that "there are comparatively few cases of hyperopia or myopia uncomplicated by astigmatism." My study showed that the refractive error of only 12 per cent of the 1260 eyes examined remained simple spheres under atropine.

A little later, while speaking of astigmatism with the rule, he said, "While this type of error is more frequent in young persons than in adults, such is not the case in senility." My findings showed that in all types of refractive errors under homatropine the astigmatism with the rule was present 586 times, while under atropine it was present 728 times. In other words, 79 per cent of the eyes in this series had their astigmatism with the rule. Dividing them into groups we find that of the hyperopic

patients 90 per cert of patients under 20 years of age, 70 per cent between the ages of 20 and 40, and 60 per cent of those under 40 years of age had astigmatism with the rule. Of the myopic, 63 per cent of the young, 62 per cent of the middle, and 60 per cent of the senile group showed the same form of astigmatism. Of the mixed astigmatism group, 74 per cent of the young, 85 per cent of the middle, and 70 per cent of the senile group were in this class. It will be seen therefore that while the percentage of the senile group is smaller, the same findings are present there as in others. In other words, astigmatism with the rule holds for the senile group in all classes of eyes.

The essayist also stated that "A cycloplegic is indicated in all patients except elderly persons with little accommodation." I cannot agree with him entirely, for in my study, while I found the lowest degree of difference in the senile group, they were numerous and in the same direction as in the younger patients. The summary here showed that of the hyperopic individuals 65 per cent of the young averaged .88 D. difference; 65 per cent of the middle aged group .50 D. difference; and 54 per cent of the senile group .42 D. difference. In the myopic, 68 per cent showed .51 D. decrease in the young; 63 per cent of the middle aged group averaged .43 D. less, and 41 per cent of the senile group .46 D. less. Of the mixed astigmatism cases, 83 per cent of the young averaged 1.15 D. difference; 56 per cent of the middle aged group, .54 D. difference; and 56 per cent of the senile group .39 D. difference, the change being either a decrease in the minus or an increase in the plus meridian. Combining those cases which showed changes in the cylinders, that is, totalling the cases that had either more or less cylinder with more sphere under atropine, we find that in the hyperopic group 52 per cent of the young, 48 per cent of the middle aged, and 37 per cent of the senile showed changes in the amount of astigmatism. In the myopic group 54 per cent of the young, 55 per cent of the middle aged, and 43 per cent of the senile showed differences. In all cases where the spheres increased the cylinders changed in 65 per cent, and remained the same in 35 per cent. In those cases where the spheres decreased, the cylinders changed in 81 per cent, and remained the same in 19 per cent. Those cases where the sphere remained the same had 55 per cent in which the cylinders changed, and 45 per cent in which they remained the same.

The most significant findings in the entire series were the cases which showed myopia under homatropine and became mixed astigmatism under atropine. I found this change in 12 per cent of all the myopic eyes in my series. It was also found that 21 per cent of all the mixed astigmatisms under homatropine showed hyperopic astigmatism under atropine.

My study showed that 62 per cent of all hyperopic meridians changed an average of .52 D. and that every sixth meridian in the hyperopic eye had an increase of .75 D. or more. Of those with myopia, 58 per cent decreased an average of .61 D., and every seventh meridian in the myopic eye had a decrease of .75 D. or more.

For practical purposes it is well to remember that in hyperopia the sphere increased and the cylinder decreased in 32.3 per cent of cases, and in myopia the sphere decreased and the cylinder increased in 31.2 per

cent of cases under atropine, over what it had been under homatropine. This may perhaps explain why the patient is more comfortable when his cylinder is under corrected in the hyperopic eye. The poorer relaxation under homatropine, allowed the finding of a higher cylinder than the eye actually has, so that when we think we are under correcting the astigmatism in such an eye by ordering less cylinder, we are really ordering a glass nearer the correct amount of astigmatism in the eye were it better relaxed under atropine.

Dr. Edward N. Robertson, Concordia, Kansas: Dr. O'Brien has presented a very clear and concise paper on the diagnosis and treatment of astigmatism and I am sure we all appreciate it. In the discussion my remarks will be confined to the cylinder retinoscopy.

In the case of a cooperative patient with a relatively high amount of astigmatism and clear media it is not a difficult matter to use the retinoscope and get accurate results; but with mixed astigmatism, especially low degrees, or a young child or an uncooperative patient, or one with media not perfectly clear—the very cases where we must have a reliable objective test—it is not so easy to solve our refractive problem by this method alone. In such cases nothing short of a thorough understanding of the meaning of the varying charges of light and shadows, as well as of the underlying optical principles, will assure success in the accurate estimation of astigmatic errors.

In looking for the astigmatic movements we do not always see a distinct band of light. In cases of astigmatic errors of two diopters we always get very distinct bands, but in the lesser strength, especially those under one diopter, we see only a movement of light, and this must always be observed carefully in the central portion of the cornea in the visual axis.

A hyperopic, or *with* movement, is more easily observed for point of reversal than a myopic, or *against* movement, and for this reason it is better in most cases to add minus spheres till the myopic astigmatism is changed to hyperopic before working out the strength and axis of the cylindrical defect in the eye.

The angle which Dr. O'Brien describes as between the axes of the inserted cylinder and the axis of the new band or movement, when the cylinder is a little off axis, is designated by Lindner as the angle of direction. This angle, as has been stated, is about 45°. In reality it is 45° plus one-half of the angle between the axis of the true astigmatic error and the axis of the slightly misplaced inserted or correcting cylinder. When we are correcting astigmatism which lies at or very near the vertical or horizontal meridian, there is little chance of error; but when the defective meridian of the eye lies at an oblique axis and in placing our correcting cylinder we miss this axis by 15°, 20° or 25°, the increased width of the angle of direction may deceive us unless we take into consideration another important point in cylinder retinoscopy, that of the appearance of equally and unequally mixed astigmatism.

Let us assume that we have an uncorrected plus astigmatism of one diopter at axis 50°. As a trial we place our correcting lens axis at 30°. A new hyperopic band or movement appears at 85°, which is 55° to the

left of our inserted cylinder, the increase of 10° being one-half of the difference between 50° and 30°. Fifty-five degrees is enough more than 45° to make us suspect that our cylinder is too strong. How are we going to decide which is the case? First, by noting the brilliancy and rate of movement of the two bands or movements of light which are at right angles to each other. If we find that the new hyperopic or *with* movement to the left of our inserted cylinder is of the same brightness and has the same speed of movement as the myopic movement at right angles to it, regardless of the size of our angle of direction, we know that our cylinder is correct as to strength.

AN EXPERIMENTAL STUDY OF DETACHMENT OF THE RETINA AND ITS SURGICAL THERAPY.*

LEO L. MAYER, M. D.,

CHICAGO.

With the advent in 1927 of Gonin's[1] report on a procedure for the cure of detached retina, interest in this subject was stimulated anew. Numerous reports of cases treated have appeared in the literature and, at times, some inference was drawn as to the etiology of the condition. Because of the time element and the rarity of cases, very little has been written on biopsied or autopsied material. Exactly what occurs when a detached retina is treated has not been thoroughly studied.

With these thoughts in mind, we began this study two years ago at Northwestern University. This must be considered a preliminary report as the investigation is still being carried on and is not conclusive.

The initial problem was to produce detachment of the retina in animals.

That this was not an original thought is attested by the extensive work of Weekers.[2] Because an experimental detachment of the retina is necessary in a study of this sort, a review of the methods and attempts at its production does not seem out of place at this time. In 1875 Chodin[3] showed that a loss of vitreous due to an incision into the sclera was often accompanied by a detachment of the retina. Rahlman,[4] in 1876, and Leber,[5] in 1886, produced retinal detachment by carefully introducing a foreign body into the vitreous or by simply injecting a concentrated solution of sodium chloride. Both Boucheron,[6] in 1883, and Simi,[7] in 1884, used a dilute solution of cantharidis which was injected between the sclera and the choroid. In 1895, Bach[8] resorted to an intra-ocular injection of sublimate to produce detachment in a rabbit's eye. Wessely,[9] in 1904, applied a capsule to the eyeball and caused a circular area of steam to be introduced into the collar of the capsule. Best,[10] in 1906, injected phloridzine to effect

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an experimental diabetes. An exudative detachment of the retina was noted between the choroid and retina. During the twenty years following little if any experimentation was published in this field. While studying ophthalmotonus experimentally in rabbits, Weekers[11] found detachment of the retina as a by-product, which eventually became more fascinating than his original investigation. By using the red heat of the cautery on the exposed sclera of the rabbit's eye he was able to produce an immediate detachment which persisted in some cases as long as five months. However, few tears or holes were found in the retina of his animals. In a recent discussion of a paper by Walter Parker,[12] Laird[13] refers to the experimental work of Sinclair. This is also mentioned in Ball's[14] textbook, but no references are given and we were at a loss to find any mention of it in a thorough review of the literature. Gonin and the men who have adhered to his views, especially Vogt[15] and Lindner,[16] insist upon the necessity of a hole or tear being in the retina, and have emphasized the fact that cure is possible only upon the localization and cauterization of this dehiscence in the retina.

Speculation as to the pathogenesis of detachment of the retina has brought forth many theories. Weekers divided these theories into three divisions, viz.:

1. Theory of Distension.—This emphasizes the fact that the majority of detachments are found in highly myopic eyes. Because of the stretching of the outer layers, the thin, loosely attached retina fails to follow in the enlargement of the globe and thus necessarily falls away.

2. Theory of Traction.—Here an alteration in the vitreous is presupposed. Because of this diseased process, bands formed in the vitreous attach themselves to the retina and gradually pull it away from its choroidal contacts.

3. Theory of Pressure Displacement.—It is the oldest conjecture, basing the hypothesis on the fact that an exudate, hemorrhage or what not, produced either in a diseased choroid or the posterior retinal layers, may cause forward bulging of the retina.

Various authorities have modified these three theories to suit their own ideas of the subject.

Gonin[17] feels that there are primary changes in the vitreous and probably the choroid, the retina weakens, a hole is formed, and the metamorphosed vitreous goes through this hole and dissects the retinal attachments.

Deutschman[18] has expressed himself along similar lines, but does not agree with Vogt,[19] who assumes the retinal tear to be the primary cause. Deutschman goes further and shows that the hole often is closed spontaneously or by igni-puncture without healing the detachment. Both Kalt[20] and Weekers hold to the old theory of pressure displacement, and both feel that their experimental work has proven this contention.

Von Graefe[21] was the first to enunciate the fact that a choroidal exudate was the cause. Leber[22] is the originator of the traction theory. He modified his original view from time to time, did not think the holes could be closed and was very pessimistic concerning cure. Kummell[23] persists in his view that all difficulties may be explained on the theory that in highly myopic eyes the retinal coat is easily subject to detachment because of the stretching of the other coats. Sourdille[24] thinks that it is an acknowledged clinical fact that there is fluid between the retinal layers. This must be evacuated either outside the globe or into the vitreous; thus the presence of a hole in the retina is no disadvantage. For further details of these theories one must refer to the original works, the many articles on detachment, of the past few years, and the reviews of the subject, one of which is the extensive summary by Stallard.[25]

In spite of the fact that Weekers had devised a method for the production of experimental detachment of the retina, which, in his hands, gave excellent results, it was our feeling that since the hole or tear had become the primary "bone of contention," a method to include this was necessary. After many trials we were able to develop a technique which satisfied our need for an artificially detached retina. The details of this method will be described later.

Our problem has divided itself into two phases: first, the pathological study of experimental detachment of the retina as such; and, secondly, the chemical and physical evaluation of the vitreous in eyes so diseased. A discussion of the former will be undertaken first. Reports in the literature on pathological examination of operated detachments are indeed meager. Weekers' description of his experimentally produced detachment in a rabbit's eye is most interesting. He reports "in a typical picture of minimum cauterization, the episclera is moderately edematous and infiltrated, the sclera at the same time shows no manifest lesion. The choroid, on the contrary, is profoundly altered. It is the site

of an enormous vascular congestion. The tumefication of the choroid is seen, making it appear as an erectile tissue. Intense congestion is found in the large vessel layer. An exudate is accumulated between the pigment layer and the retina."

Amsler[26] demonstrated the pathology in a case complicated by intraocular hemorrhage following cautery for a detached retina, but to our mind this complication certainly influences the picture, and thus the interesting findings are of little value.

The first attempt to study experimentally the effect of cautery on the eye is the very recent preliminary report of Luntz,[27] from Lindner's clinic. He was interested in knowing whether the white or red heat of the cautery was most satisfactory. Retinæ of rabbits exposed to the cautery for various lengths of time, from two to twenty seconds, were removed after five to twenty-two days and studied. Luntz felt that it was of importance to note that tissues not in the immediate vicinity were affected by the cautery, in spite of Wessely's[28] comment to the contrary. In the discussion of this report, Lindner[29] brings out the point that dire effects may be toxic in nature, and are found especially where the cautery is used for longer periods of time.

Of more recent publication is Guist's[30] work on his new operation with the potassium hydroxide stick. Sections showing the pathology resulting after one second, which Guist found to be the optimum time, accompany the article. Lindner[31] feels that this new method, because of its chemical nature, approximating the method of Sourdille with oxycyanide of mercury, does less harm to the tissues than the cautery. A pathological study of Guist's sections would seem to bear out this statement.

After many attempts with varying procedures, the simple technique for producing a mechanical detachment of the retina may be described as follows: The rabbit is placed under ether anesthesia and the usual operative methods to prevent infection are carried out. The eyeball is rotated upward as far as possible, the conjunctiva incised down to the sclera. A small incision, from 1½ to 2 mm., is made with a cataract knife through sclera, choroid and retina, so that the vitreous is not entered, or at least very little disturbed. A small thin spatula, about 1 mm. wide, is inserted between the retinal and choroidal coats, and by a backward sweeping motion the layers are separated. A small amount of vitreous usually escapes. No attempt is made to close the scleral opening, as this is the permanent hole which reaches through into

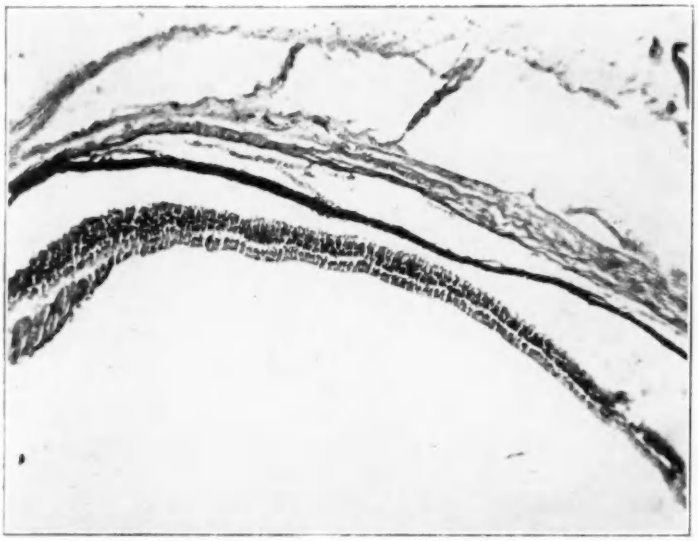

Fig. 1. A section of a normal rabbit's eye showing the separation of layers due to defects in the usual laboratory methods. (×45.)

Fig. 2. The layers separated mechanically in the production of experimental detachment of the retina. (×45.)

the vitreous. The conjunctiva is closed with silk sutures, atropin and 2 per cent mercurochrome instilled, and no dressing applied. The entire procedure may be checked with the ophthalmoscope. As was stated previously, many attempts have made this method the one of choice, and an explanation of why this method was accepted would simply be presuming on your time and patience. Suffice it to say that ether caused least loss of animals, mercurochrome prevented loss of eyes by infection, and drawing off vitreous previous to opening the chamber did not enhance the ultimate results. Fig. 1 demonstrates the normal rabbit's eye in a microphotograph. Fig. 2 is that of an artificially detached retina according to our method.

In order to know the effects of operative handling on detached retina, we thought it appropriate to study the pathology of the various operative agents on the normal eye. Fig. 3 is the normal rabbit's eye after using the actual cautery. Fig. 4 shows the results with the Paquelin cautery, the method of Gonin. In Fig. 5, Larsson's[22] method of using the diathermy ball is depicted. Fig. 6 is an eye removed after intra-ocular diathermy with a fine needle. Fig. 7 shows the Sourdille method with cyanide of mercury. Fig. 8 shows the results according to the most recent method of Guist[20] with potassium hydroxide stick. These same procedures were used on our artificially detached retina and are demonstrated by Figs. 9 to 14.

Fig. 9 is a detached retina treated by actual cautery for ten seconds.

Fig. 10 is a detached retina treated with the Paquelin for two seconds.

Fig. 11 is a detached retina treated with the diathermy ball for five seconds.

Fig. 12 is a detached retina treated with the diathermy needle for two seconds.

Fig. 13 is a detached retina treated with cyanide of mercury.

Fig. 14 is a detached retina treated with the KOH stick for one second.

Reattachment seems to have taken place in all procedures by producing an adhesive exudate between choroid and retina. A detailed description of the pathological findings in our study, because of the many methods used, would be presumptuous. Therefore only brief statements of the changes are included. The actual cautery and the Paquelin caused a large hole to be formed in all

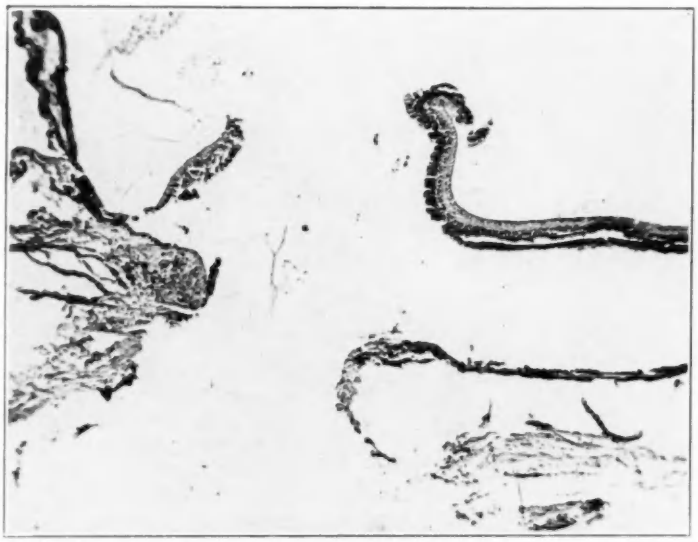

Fig. 3. Destruction in the normal rabbit's eye caused by the actual cautery used for 5 seconds. Complete healing has not been allowed to take place. (×45.)

Fig. 4. Destruction in the normal rabbit's eye due to the Paquelin cautery (Gonin's method) used for 5 seconds. Removal after 5 days. (×45.)

Fig. 5. Pathology caused by the use of the diathermy ball (Larsson's method) on the intact rabbit's eye. (×45.)

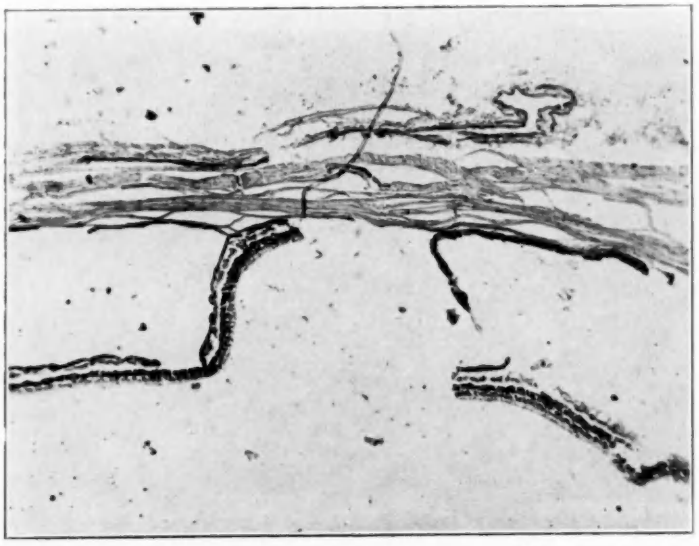

Fig. 6. Destruction caused by use of diathermy needle after 5 seconds in intact rabbit's eye. Removed after two weeks. (×45.)

the layers with which they came in contact, regardless of the time of exposure. In all procedures, with the exception of Guist's, the area affected did not extend more than one-half centimeter in radius from the point of contact. With the hydroxide stick there is a tendency for the chemical to spread, especially if the time of application is for longer than one second, and thus a larger area may be included. In our hands, neutralization with such a mild acid as acetic does not mitigate against this defect. The Sourdille procedure with oxcyanide of mercury had little or no effect on the normal eye, and it would seem that the sclerotomy is the important part of this method.

Larsson's diathermy ball causes no destruction of tissue except for the pouring out of an exudate. Because of its harmless effects it would seem to be a rational procedure if the electrical factors were standardized. The diathermy needle must be used with care. In the absence of fluid actual cauterization of the tissues takes place. Where spontaneous reattachment occurred after mechanical detachment we are unable to say at present just what becomes of the retinal holes, as pathological study did not reveal this.

That spontaneous reattachment might occur even without therapy was not disregarded. However, in watching these cases ophthalmoscopically over long periods of time, no reattachment occurred within the first three weeks, and operative methods were attempted so that actual healing was ended before this period. Spontaneous reattachment did occur in all cases after six weeks in spite of a remaining hole in the retina.

This brings us to the second phase of this work. The startling fact that spontaneous reattachment occurred regardless of the hole in the retina after a period of six weeks led us to try other methods. It has been known for a long time that detached retina occurs often in a soft eye. Also, in those highly myopic eyes which seem more predisposed to this condition, the existence of what is called "fluid vitreous" has always been a questionable quantity. Redslob and Reiss[33] have demonstrated conclusively that changes in the pH of the intraocular fluids play an important and direct rôle in the maintenance of intra-ocular tension. If the pH is on the acid side the tension tends to be lowered, while the alkalin ion raises this factor. We tried various dilutions of acetic and hydrochloric acid, and while we were able to produce a detachment by this method we found it beyond control. An acid strong enough to cause the detachment went further

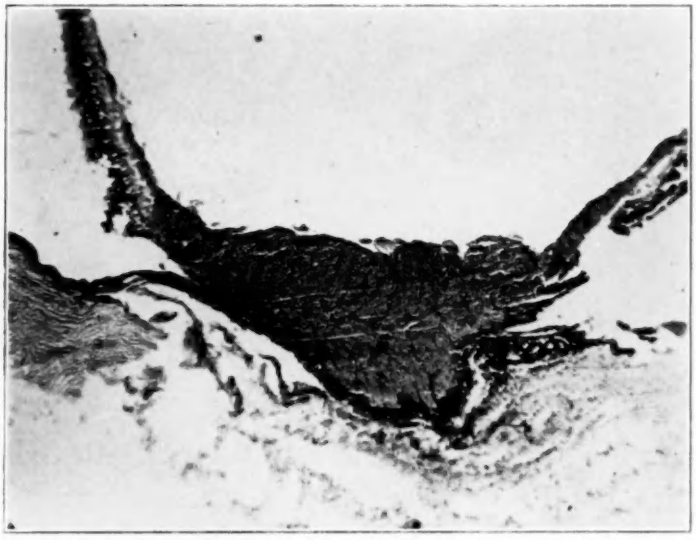

Fig. 7. The intact rabbit's eye after use of cyanide of mercury injection (Sourdille's method). (×45.)

Fig. 8. Use of KOH stick (Guist's method) in normal rabbit's eye after 3 seconds. Complete healing has not taken place. Removal after 4 days. (×15.5.)

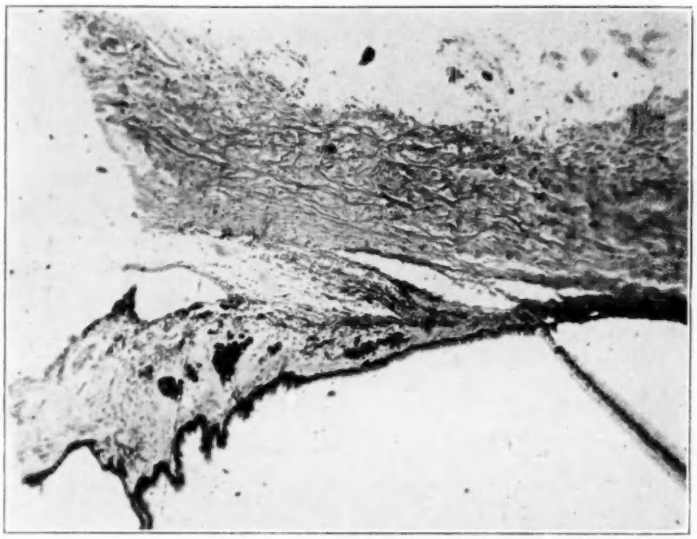

Fig. 9. Mechanically detached retina treated by actual cautery for 10 seconds. Removal after 3 weeks. (×45.)

Fig. 10. Mechanically detached retina treated with Paquelin cautery for 2 seconds. Removal after 5 days. (×15.5.)

and disintegrated the retina and choroid. If the acid was not of a certain maximal strength no detachment occurred. Finally trypsin was tried. In stock concentrations digestion took place after detachment. However, by diluting the trypsin a detachment occurred without destruction of the layers and remained over a long period of time. We were greatly interested to know just what changes in the make-up of the vitreous took place, and Chart 1 shows a comparison of the various vitreous elements in the normal rabbit's vitreous and in one treated with trypsin.

CHART 1.

	Normal	Trypsinated
Surface tension[34]	52.7	56.84
Sugar[35]	49.0	81.9
Total nitrogen[36]	26.3	113.2
Amino-nitrogen[37]	8.16	5.45

As the surface tension of water is 70.7, it is easily realized that the fluidity of the vitreous is increased by trypsin. Also sugar and total nitrogen in mm. per 100 cc. are enhanced. The method of producing these results was as follows: A stock trypsin powder was mixed with physiological saline to form a supersaturated solution. This was sterilized by putting it through a Berkefeld filter. The filtrate was then tested for digestive potency in various dilutions with carmine fibrin. Three minims of a 1 to 4 dilution were injected with a small hypodermic into the vitreous chamber of rabbits' eyes. This was recovered by removal of the globes after five days and analyzed. Comparison was made with normal controls. A detached retina so produced has been found ophthalmoscopically after a period of five months, in spite of the fact that there were no holes in the retina, the hole made by the injecting needle being entirely closed in about one week's time. In Baurmann's[38] study of the vitreous, he has expressed the view that the vitreous plays a passive rôle in detachment, that tears or holes are secondary only but prevent reattachment. Because of these views Baurmann has been the chief exponent of the so-called hydrostatic theory. Duke-Elder's extensive investigations are known to all of you, and in one of his latest publications[39] he expresses the view that if "one has a fluid vitreous that gives no real support to the retina from within, and then the eye is made the object of a trauma, here are all the materials at hand for caus-

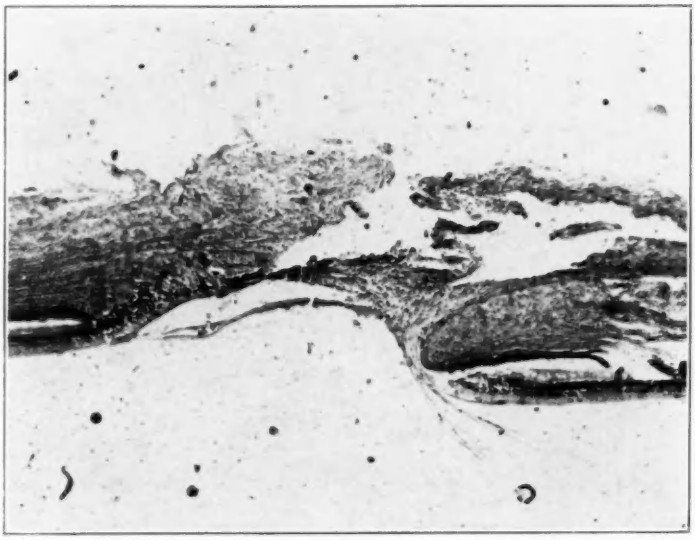

Fig. 11. Mechanically detached retina treated with diathermy ball for 5 seconds. Removal after 5 days. (×45.)

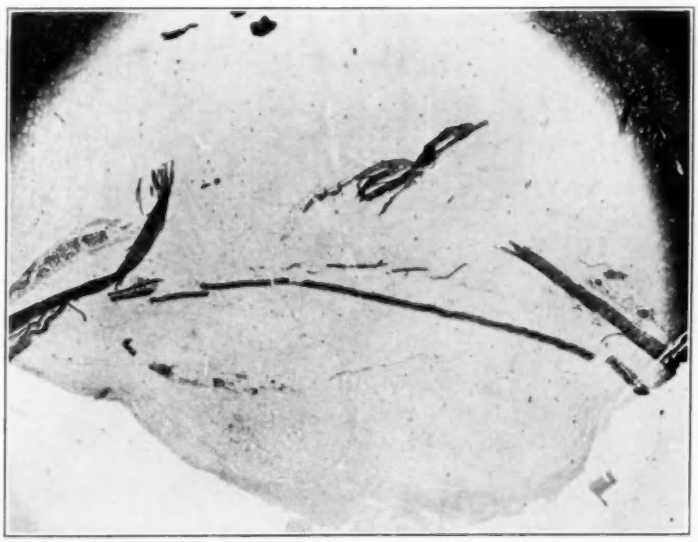

Fig. 12. Mechanically detached retina treated with diathermy needle for 2 seconds. Removal after 5 days. (×10.)

Fig. 13. Mechanically detached retina treated by Sourdille's method. Removal after 5 days. (×40.)

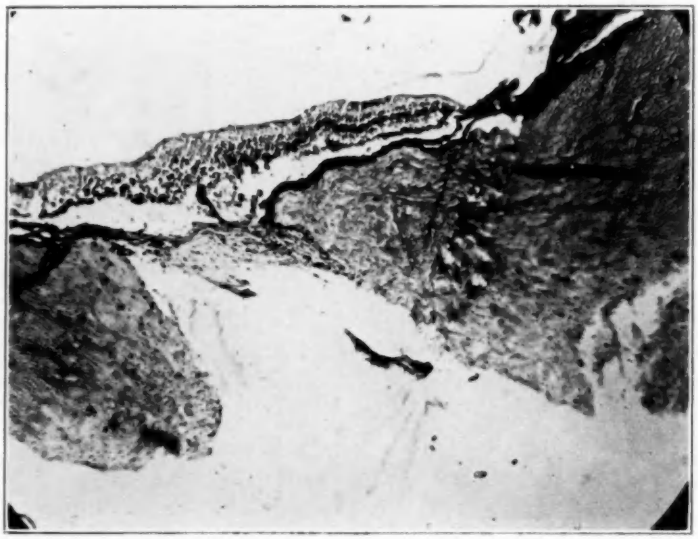

Fig. 14. Mechanically detached retina treated with KOH stick for 1 second. Removal after 5 days. (×40.)

ing a detachment of the retina." Mention should also be made at this time of Anderson's book on detachment of the retina which has just come from the press. It reviews the entire literature up to 1930. It is interesting to note that Anderson[40] had the same difficulty we had in trying to reproduce Weekers' colmatage. Also, a verbatim quotation[41] seems important. "It can be concluded that, though the results of ignipuncture are beyond doubt, one is not qualified yet in assuming that in all cases the tear was instrumental in producing the detachment now cured by the operation which has closed it."

The pathological study of the effects produced when chemical agents or enzymes are introduced into the vitreous, to cause a retinal detachment, has not been completed. That the trypsin effect may not be entirely local on the vitreous itself but rather a general disturbance of body fluids, may be suspected, as brought out by Buckley,[42] who has shown interesting results of subcutaneous trypsin injections in relation to the blood sugar.

IMPRESSIONS.

1. From our study, it would seem rational to deduce the fact that the retinal tear or hole is not the entire factor in maintaining a detached retina.

2. Changes in vitreous composition due to acids or enzymic alteration which cause a detachment of the retina, point to another factor in this condition.

3. Permanent experimental detachment of the retina has not been produced, and further progress in a study of this kind must await such a procedure.

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DISCUSSION.

DR. A. L. BROWN, Cincinnati, Ohio: Those concerned with so called spontaneous retinal detachment are interested in the pathogenesis, the best curative measure, and the rôle of tears.

The mechanical theories are the oldest and most obvious. This is doubtless because the condition primarily presents such a strikingly physical appearance and so often occurs in persons apparently in good health. Gonin's recent work and his emphasis on the importance of tears has stimulated the correlation of the work of other observers to the point where anatomic theories no longer explain many facts. A certain number of detachments in highly myopic eyes may be due primarily to a receding of support from the vitreous. Those eyes in which no detachment occurs may be filled with a more fluid vitreous or sufficient amount which follows the change in the shape of the globe enough to form a support.

Gonin describes the blackish-gray opacities in front of the posterior pole of the eye which he believes corresponds to the posterior surface of the vitreous body detached and retracted from behind forward. These he has seen in many myopic eyes with and without detachment. If a detached vitreous may be present in both instances some other factor must enter to complete the conditions for retinal detachment.

Last year, impressed by the weight of Gonin's many observations, I felt that the basis of cause and cure rested on the tears. I am not so sure now for I have had three more cases which responded to the cautery, although two of them revealed no tears even after prolonged bed rest. Lasting improvment was effected by introducing the cautery at the point of greatest detachment. Van Lint (Arch. d'Ophth., September, 1930) maintains that cautery puncture induces adhesion between the retina and sclera causing direct mechanical reattachment. Other observers have likewise felt that undue importance has been given to the tear as to its relation to the cause and cure by cautery.

The essayist has evaluated existing opinions and has proceeded with experimental investigations. His most significant disclosure is failure to produce a permanent detachment. This at first thought seems rather strange, considering the usual clinical prognosis. It seems that experimental mechanical detachment with the factors in the author's hands does not coincide with clinical cases. Even the areas of old choroidal atrophy, many formed and discrete vitreous opacities, with retinal exudates, and hemorrhages, so often described, do not complete the picture, for the same conditions may hold in so many cases without detachment. Beyond these physical manifestations there probably lies marked biochemical changes in the retina and adjacent structures and a variety of combinations may be necessary to produce an actual detachment.

A consensus of opinion at the present seems to indicate that thermopuncture is the most effective treatment. The action of chemicals, especially strong caustics such as sodium hydroxide, might be more difficult to control, especially in an already disturbed eye, than a cautery whose sole action is by heat. Diathermy introduces the element of conductivity involving other ocular structures, whereas it is probably wiser to limit the field of action.

Dr. Sanford R. Gifford, Chicago: I just want to say that the sections Dr. Mayer showed were some that had been embedded in paraffine, and that certain changes in the sclera were in all probability due to the embedding material and not the result of cauterization.

I think the interesting thing about this work is the continuous reattachment when the hole is still present. I hope Doctor Mayer will go on with his study of the vitreous and perhaps be able to tell us more about it after he has finished his work.

Dr. Leo L. Mayer, closing: I want to take this opportunity to thank Dr. Gifford and others of the Northwestern University for their help in this work.

ANGIOMATOSIS RETINÆ WITH CEREBELLAR CYST (LINDAU'S DISEASE).

WALTER S. ATKINSON, M. D.,

WATERTOWN, N. Y.

Since Lindau[1,2] correlated the syndrome of hemangiomas of the cerebellum and retina, 1926-1927, and Cushing and Bailey[3] so recently reviewed the literature when reporting their case in 1928, to include a review here seems unnecessary.

That the association of angiomas of the retina with angiomatous and cystic lesions in other parts of the body, particularly with a cerebellar cyst and its familial tendency, is not generally recognized by ophthalmologists, is evident by the statement of Cushing and Bailey[3] that "out of many examinations during the patient's life by at least eight observers, the presence of the angioma was not detected."

The large tortuous retinal vein had been observed, but its significance evidently not appreciated.

Cushing and Bailey,[3] in reporting their case, state that so far as they are aware it is the first in which the histological diagnosis of the cerebellar lesion has been verified during the life of the patient, which adds interest to the patient's history, to be given below, who is still alive and well.

J. R., aged 32 years, paper finisher. He was first seen by me November 27, 1926. His chief complaint was failing vision, particularly in the right eye, for the past nine months. He also complained of very severe occipital headaches for the past few months and pain in the back of the neck, occurring more frequently during the past month and worse at night, so severe some days that he was obliged to remain in bed.

For a few days prior to the examination he had felt dizzy in the afternoon. The dizziness was sometimes accompanied by nausea, but he rarely vomited. In walking he had a tendency to bear to the right but had never fallen.

In 1919, on discharge from the army, he first discovered that he could not see as well with the right eye as with the left.

At the age of six years he fell from a tree, striking his head and was in bed one week. There is no history of other members of the family being similarly affected, but one sister is in the State Hospital for the insane. The hospital reports that nothing pathological was found in her eyes.

Two children died shortly after birth; cause unknown.

Examination of the eyes: Right eye, vision 10/200, with correction 20/100; left eye, vision 20/25—1, with correction 20/20. Eyes quiet. Pupils equal and active to light and accommodation. No nystagmus, associated movements normal and otherwise eyes appear normal. Peripheral fields normal.

Ophthalmoscopic examination: Right eye, media were clear; disc margin quite blurred, more above and temporally, and the retina surrounding the disc edematous. About two disc diameters up and temporally from the disc were two small, irregular, whitish patches about the size of the disc, only elongated, apparently retinal exudate. There was a similar smaller patch quite near the disc, up and nasally. The superior temporal artery was greatly enlarged, at least three times its normal size, with many aneurysmal swellings along its course, varying in size, some twice the diameter of the enlarged artery, and on a terminal branch, pretty well in the periphery, were several quite large aneurysms as compared with the small branches of the artery.

The superior temporal vein was very much the same color as the artery but considerably larger, and on following it toward the periphery it increased enormously in size, becoming more and more tortuous until in the periphery it disappeared in a vascular tumor mass having the appearance of a tangled coil of enlarged vessels.

The other retinal vessels appeared quite normal except that possibly the veins were slightly enlarged.

Left eye: The media were clear, disc normal, and the veins a little full. There was a large, irregular, pigmented, whitish, chorio-retinal patch extending vertically between the superior and inferior temporal retinal vessels, about two disc diameters to the temporal side of the macula lutea and about one disc diameter in width, with a fringe of pigment around the edge.

On November 29, 1926, the patient was admitted to the House of the Good Samaritan, Watertown, for observation.

Summary of general physical findings:

His temperature was 98°, the pulse 80 and respirations 20. He was suffering acutely with occipital pain and the movement of his head brought on a paroxysm of pain. The cranial nerves were apparently not impaired. The reflex of the right biceps was more active than the left. The left knee jerk was a little more active than the right but both were exaggerated. The ankle jerks were sluggish but the right more active than the left.

After admission to the hospital, he had to be catheterized. This had never been necessary prior to admission. His urine showed a faint trace of albumen, but was otherwise normal. The blood and spinal fluid Wassermann was negative. The blood count and blood chemistry were normal. The cerebrospinal fluid was clear with the pressure greatly increased, but the laboratory findings were normal.

Ear, nose and throat examinations were reported normal except for a chronic inflammation of the left antrum.

A provisional diagnosis of cerebellar tumor was made, and the patient sent to St. Luke's Hospital, New York City, where he was admitted December 9th.

The report from St. Luke's Hospital added the following: The right pupil was smaller than the left, both were irregular and reacted sluggishly to light and accommodation. There was nystagmus to the left but no strabismus.

Ophthalmoscopic examination showed a choked disc in the right eye and an optic atrophy in the left eye. (No note was made of the angiomatous condition of the right eye.) Both tympanic membranes were dull and slightly injected.

Neurological examination indicated weakness of frontalis muscles. The biceps and triceps were active and equal, and the left knee jerk exaggerated. There was a suggestive Gordon and Babinski.

A decompression operation was done, exposing the cerebellum. A cystic tumor was found protruding from below the lower surface of the left cerebellar lobe and pushed downward through the foramen magnum toward the midline. It contained quite clear fluid (5 to 8 cc.), and the top of the cyst was cut off to be used as a pathological specimen.

Macroscopical examination: The specimen consists of a small piece of fibrous looking tissue which on section appears hemorrhagic.

Microscopical examination: Sections of the wall of the cyst show a very small amount of cerebellar tissue (microphotographs 1 and 2), the cells being rather disorganized, small masses of glia infiltrated with blood and the more solid portion of the tissue composed of a large number of dilated vascular sinuses embedded in a glial reticulum in which are a number of rather large clear cells. These cells are probably also glial in origin, having a small

Fig. 1.

compact, deeply staining nucleus. Some of the vascular sinuses contain many polynuclear cells, apparently early thrombi forming in the wall. The lining of the cyst is apparently glial only. None of this tissue appears to be neoplastic, and many of the changes are inflammatory, due to pressure or trauma.

On January 7, 1927, he was discharged from St. Luke's Hospital, improved. Dr. L. C. Knox, pathologist at St. Luke's Hos-

pital, very kindly reviewed the slides of the cyst wall removed at the time of operation, finding tiny angiomatous areas, and states that unquestionably the cyst is one of those arising from and in connection with an angioma, as shown in the accompanying microphotographs.

On December 10, 1927, when next seen by me after the operation, he was having practically no headaches and felt well, but

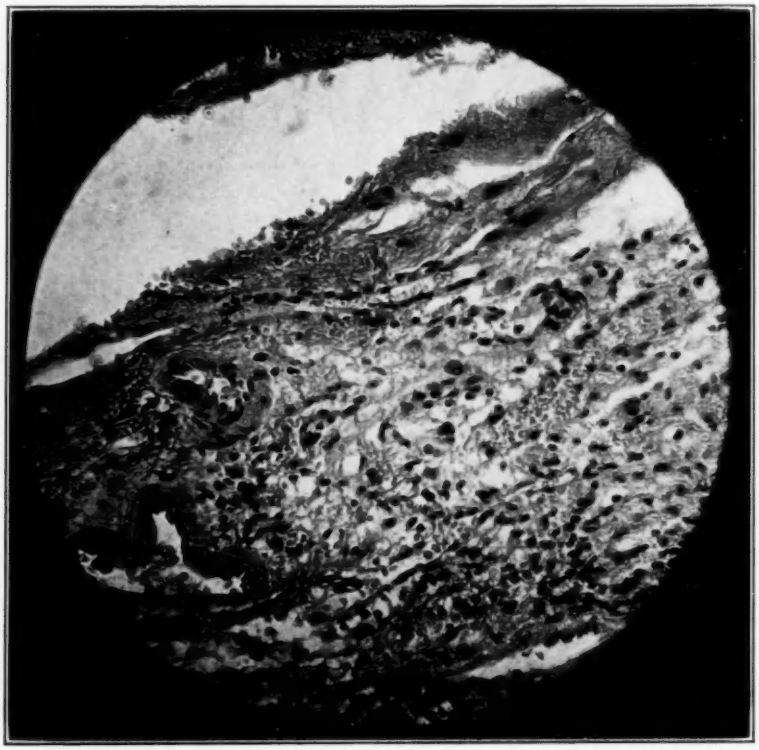

Fig. 2.

said that he has been unable to see with the right eye since the operation.

Vision: Right eye, hand movements temporally; left eye, 20/50 and with correction 20/20—3. The loss of vision in the right eye was due to a large detachment of the retina, otherwise the eyes were about the same.

On June 19, 1930, there was no light perception in the right eye; in the left eye, the vision was 20/40 and with correction 20/20. The detachment of the retina in the right eye had so increased that only a very little of the enlarged vessels could be seen, as shown in the accompanying photograph which Dr. Arthur J. Bedell kindly took at this time. On January 30, 1931, he wrote saying that he was working and felt well.

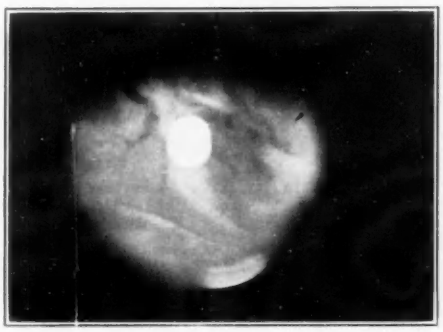

Fig. 3.

SUMMARY.

Male patient, aged 32 years. (This corresponds with the sex and age of other cases reported.)

The enormous vein with angiomatous tumor mass in the periphery, white patches of exudate in the retina and the enlarged artery with aneurysms presents a rather typical picture of angiomatosis retinæ very often referred to as von Hippel's disease. This, together with the cerebellar cyst with angiomatous areas in the cyst wall seems sufficient to classify this as a case of Lindau's disease.

That no other members of the patient's family have been found to have this disease does not preclude it; also, Möller,[4] in a recent communication regarding Lindau's disease, states: "There should be a cerebral tumor plus familial incidence, or retinal angiomatosis plus cerebral tumor, with or without familial tendency to make a diagnosis of Lindau's disease."

This report adds a second case of Lindau's disease where the histological diagnosis of the cerebellar lesion has been verified during the life of the patient.

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DISCUSSION.

Dr. Arthur J. Bedell, Albany, New York: In the last two years I have seen six cases of angiomatosis retinæ. Three of these were referred to in an article published in the American Journal of Ophthalmology, May, 1931. The literature was completely reviewed at that time.

The most spectacular finding in all reported cases, as well as those I have seen, was an extremely large vessel or vessels. Frequently this was an immense vein, so that in every case in which great vessel fullness is present the periphery of the fundus must be searched for the characteristic angioma. This may not be present for a considerable time, even many months, after a probable diagnosis has been made. The angiomas vary greatly in size and are often mistaken for retinal hemorrhages. The entering artery and draining vein can, however, be distinguished.

(Dr. Bedell presented lantern slides of many of the historical cases and a photograph of one previously unpublished.)

Dr. George F. Suker, Chicago: I wish to put on record an unusual case of angiomatosis retinæ in a soldier, 36 years of age, whose family and personal history were negative. The patient was also serologically and neurologically negative. He was sent from army hospital to army hospital, and finally landed at the Edward Hines, Jr. Hospital (veterans' hospital). No diagnosis had been made at the other hospitals. He showed no symptoms whatever except a reduction of vision in the right eye. (See photographs.)

In the retina of the right eye the superior nasal vein was exceedingly dilated and at points constricted—in other words, kinked. The accompanying artery was likewise dilated, but not kinked. There was a retinal detachment at the site of the elevation of the vessels, for these vessels did protrude into the vitreous and on their apex the retina was elevated or detached, if not edematous. There was a sclerotic belt in the choroid following the course of these vessels. Both artery and vein abruptly disappeared in a "lake" of blood in the extreme upper nasal quadrant of the retina. Seemingly, there was an anastomosis at the very point of disappearance. No view was obtainable of the fundus beyond this lake. The external eye in this area was normal. However, transillumination at this point showed this lake. There were numerous elevations and distortions of accompanying vessel branches. The disc was normal excepting on

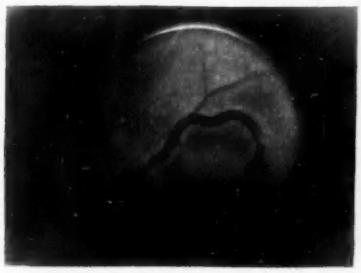

Fig. 1.

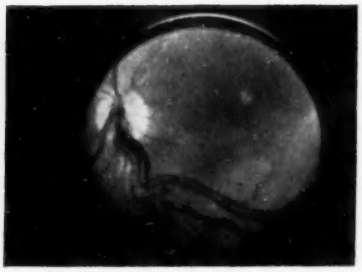

Fig. 2.

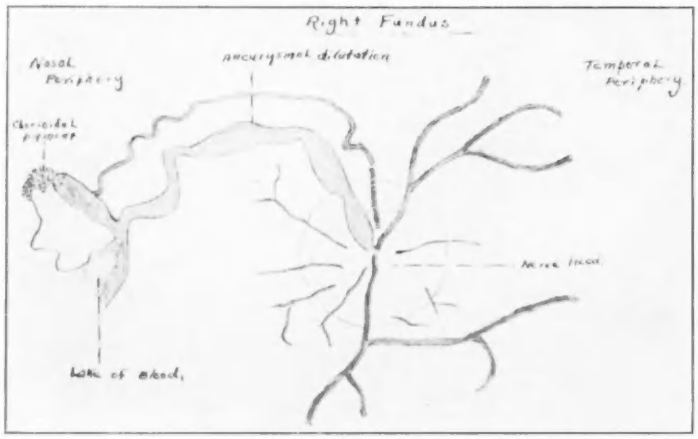

Fig. 3.

nasal half, where the retinal vein and artery turn upward to the nasal field. This portion of the disc was deeply cupped.

The point in question is, how long do we have to wait before we have cerebral symptoms in this case? I have gone carefully into his family history and cannot find any member on either side with an eye lesion or brain symptoms which in any way could be likened to this particular lesion. May it not be that instead of having a cerebral lesion accompanying this eye angiomatosis, we may now and then have such a lesion purely limited to the retinal vessels themselves? If this idea is correct, then I believe this is the first case of its kind to be reported, and therefore put it on record.

If we can limit our idea of angiomatosis to ocular lesions *per se*, then we have a large field, and I think these will be more common than we think at present. I heartily agree with Dr. Bedell that in years gone by there were many large, distorted vessels that emptied into a lake of blood that we did not recognize as angiomatosis. We looked upon them

as hyperplasia of the vessel walls, but not tumor formation. Whether or not I am right depends upon the pathological examination of this case.

In conclusion, it is suggested that we should be very careful in examining these fundi not to lose sight of the fact that they may have, in addition to von Hipple's disease, a pernicious anemia, sometimes a polycythemia, both of which may cause a marked dilatation and corrugation of the vessels. It is further suggested that such suspicious cases should have a careful blood examination made. One was made in this instance and perfectly normal blood was found.

Dr. Walter B. Atkinson, closing: I would like to thank Dr. Bedell and Dr. Suker for their very kind discussion.

Dr. Bedell did not mention in his discussion and presentation of the pictures of angiomatosis retinæ whether or not any of the patients had angiomatous areas elsewhere in the body, which was really the point of the paper—not simply a case of angiomatosis retinæ, but one of Lindau's disease.

VISUAL FIELDS IN CHRONIC GLAUCOMA—THE EFFECT OF REDUCED ILLUMINATION.

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The degree of success of treatment, whether medical or surgical, is well known to be dependent upon early diagnosis in all fields of medical endeavor. This is particularly true of the chronic forms of glaucoma and has stimulated the development of reliable methods for early diagnosis and noting the progress of the disease. The importance of careful measurement of the field of vision in these cases has long been recognized. Chronic glaucoma is more dependent upon perimetry for its understanding than perhaps any other disease, and many advances in this method of examination have been associated with its study.

The most important addition to the methods of visual field examination has been the introduction of the tangent screen and the use of small test objects for the investigation of the central portion of the field. Many different kinds of screen have been developed, varying in size and shape and in the methods of recording the defects uncovered. The screen devised by Thomasson is simple, convenient and perhaps more adaptable to office and clinic use than any of the larger screens. Its chief disadvantage, however, is the limitation of its use to scotometry, as it is not sufficiently large to permit mapping the field of a 1 mm. object at a distance of 2 m. Nevertheless, it has served well the purpose for which it was devised and has probably stimulated the careful investigation of the central field, especially in the early diagnosis of glaucoma.

The use of a tangent screen at 1 or 2 m. with small-sized test objects has the effect of magnifying such defects as may be present, or, to use the language of Ferree and Rand, of increasing the sensitivity of the test. As these authors have pointed out, there are three ways in which this can be done. The first is exemplified by the present method of using the tangent screen. A second has to do with the difference between the object and its background. This factor they have incorporated in a perimeter

of their own design. The third variable concerns the illumination. Combinations of these offer further possibilities.

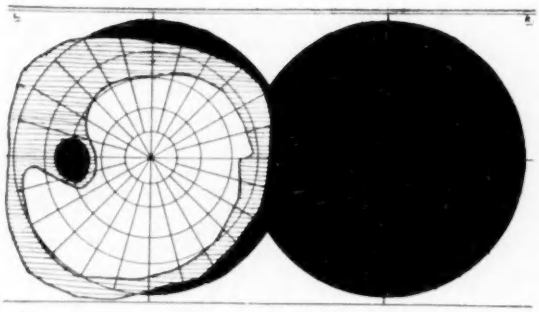
It is the purpose of this paper to report the effect of the combination of the first and third in a small series of cases. The method used, although not scientifically controlled in a strict sense, seems to have very definite value in spite of its crudeness. It is described because it reduces to a minimum additional apparatus and expense and can be made use of without adding greatly to the time consumed.

In all cases Thomasson's screen was used at a distance of 1 m. and a test object of 1.5 or 1 mm. After adjusting the patient the lighting was reduced on the screen, by closing window curtains, to an average of about 0.20 feet candles, as measured with an illuminometer. After allowing for adaptation to take place, the 1/1000 field was outlined, repeated tests being made to avoid error. After this had been completed the curtains were widely opened and the tests repeated, the blind spot being carefully mapped and the search for scotomata carried out. When these tests were first carried out, immediately after the patient left, as a control a normal person was examined under identical conditions.

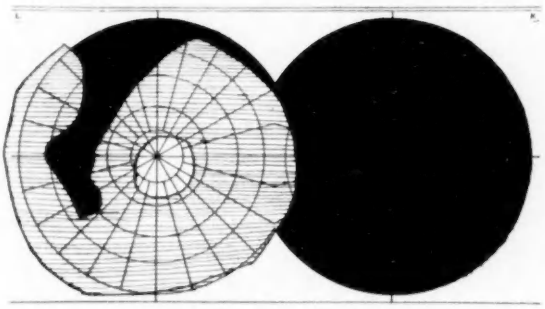
Chronic glaucoma seems an especially appropriate disease for which this procedure can be used. The work of Derby and his collaborators has clearly demonstrated that a lowering of the light minimum is probably the earliest detectable change in incipient cases. This being true, one should be able to detect earlier or more advanced field changes in these cases by reducing the illumination. Furthermore, progress of the disease could be more surely demonstrated.

With these considerations in mind, the fields of eleven cases of chronic glaucoma watched over varying periods of time are presented.

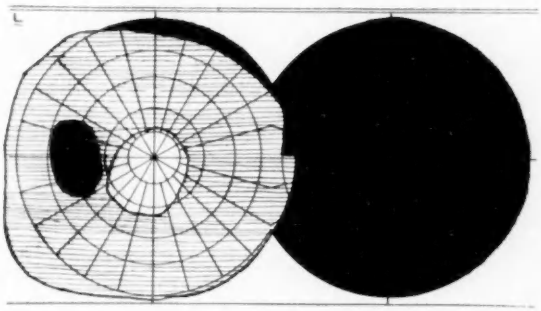
Six charts of case 1 are presented. This patient has been under observation since September, 1923, and has had repeated field studies made over a period of more than seven years. She is, therefore, well trained and an accurate observer. Many attempts to discover scotomata or other changes in the central field and about the blind spot have always been unproductive. Her right eye was blind when first seen. The first chart presents a normal blind spot and the field for a 1.5/1000 test with full illumination.



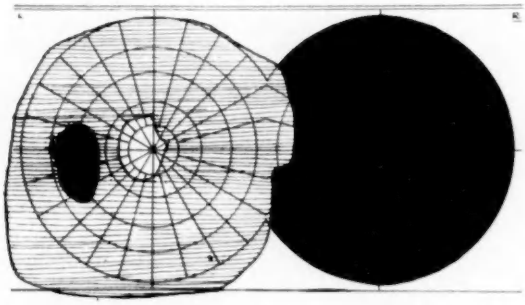
Case 1—Mrs. E. C., A-1115. Chart I—Feb. 18, 1929, 9:30 a. m., sun out; vision, 6/9+; tension, 2/5.5=29 mm.; 1.5/1000 outer field, good light; blind spot, good light; inner field, dim light.



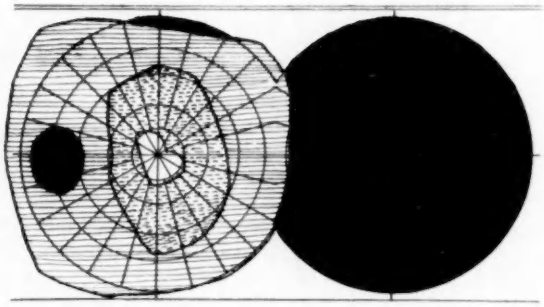
Case 1—Mrs. E. C., A-1115. Chart II—Feb. 12, 1930, 9:30 a. m., sun out; vision 6/8+; tension, 3/5.5=25 mm.; 1.5/1000 outer field, good light; blind spot, good light; inner field, dim light.



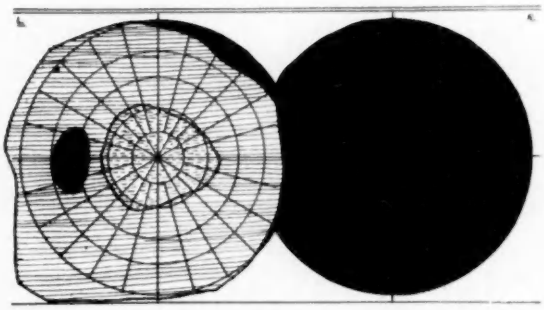
Case 1—Mrs. E. C., A-1115. Chart III—July 30, 1930, 10:30 a. m., bright, cloudy; vision, 6/8+; tension, 3/5.5=25 mm.; 1/1000 outer field, good light; blind spot, good light; inner field, dim light.



Case 1—Mrs. E. C., A-1115. Chart IV—Jan. 20, 1931, 10:30 a. m., bright, cloudy; vision, 6/8+; tension, 4/7.5=31 mm.; 1/1000 outer field, good light; blind spot, good light; inner field, dim light.



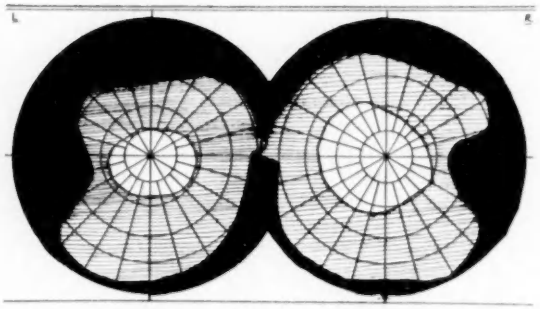
Case 1—Mrs. E. C., A-1115. Chart V—Jan. 30, 1931, 2 p. m., dull, snowy; vision, 6/8+; tension, 3/5.5=25 mm.; 1/1000 outer field and blind spot, good light; 2/1000 middle field, dim light; 1/1000 inner field, dim light.



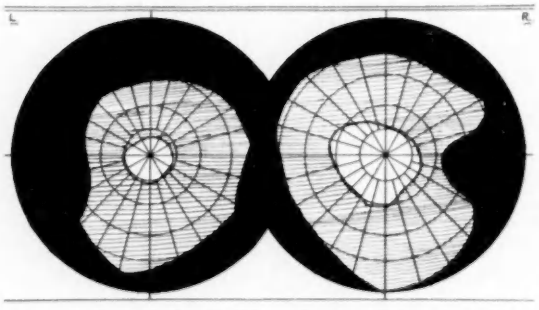
Case 1—Mrs. E. C., A-1115. Chart VI—Feb. 14, 1931, bright, cloudy; vision, 6/6—; tension, 4/5.5=24 mm.; outer field and blind spot 1/1000, good light; inner field, 2/1000, dim light; 1/1000 not visible, dim light.

The shaded area represents the reduction of the field when the light was reduced. Chart II, taken one year later, shows the only scotoma ever found in this case connected with the blind spot. Except for this area the 1.5/1000 field shows practically no change. With reduced illumination there is a marked contraction of the 1.5/1000 field as compared with the previous chart. The conditions of the test are practically the same as regards the time and brightness of the day. In Chart III the chief changes are represented by a definite enlargement of the blind spot and the nasal step. In Chart IV, six months later, the blind spot remains enlarged and there is little change in the 1/1000 field with good illumination. There is, however, a further reduction of the 1/1000 field with reduced lighting. Further change is noted in the fifth chart in which a 2/1000 field is plotted for the first time with reduced light. In the last chart (VI) the 1 mm. object is no longer visible when the light is reduced and the 2/1000 field has undergone still further contraction. Throughout all these observations it will be noted that there has been relatively little change in the extent of the 1/1000 field when good lighting is used. There has been some variation in the blind spot but no scotoma has ever been discovered, with the exception noted, although a careful search has been made. The striking thing about these charts, then, is the gradual reduction of the field in a dim light without appreciable change in good light. This sequence of events corresponds well with other clinical evidence of the advance of the process. The patient has been having more subjective symptoms lately, the tension has been more difficult to control with miotics, and some surgical intervention seems imminent in the future in spite of the fact that the patient is a poor surgical risk.

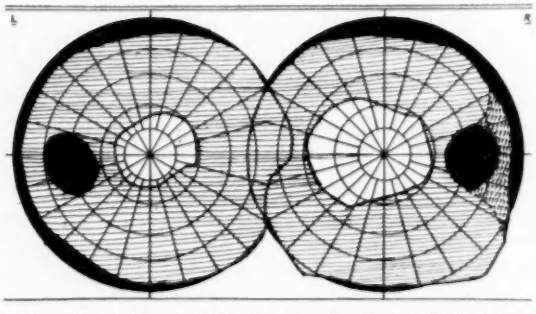
Five charts show the progress of the disease in case 2. There is a greater degree of variation in this case, but it seems fair to conclude that the fields in reduced illumination show the progress of the disease more clearly and are more consistent with the other clinical features of the case than those taken with good light. This is not a case of primary simple glaucoma, for the slit-lamp revealed the presence of very fine deposits diffusely scattered over the posterior surface of the cornea. The change shown in Charts VII and VIII consists mainly in a slight reduction in the 1.5/1000 field when the lighting was reduced. A marked change is to be noted in Chart IX. If one were to consider the fields taken with good light only, a very marked improvement would be thought



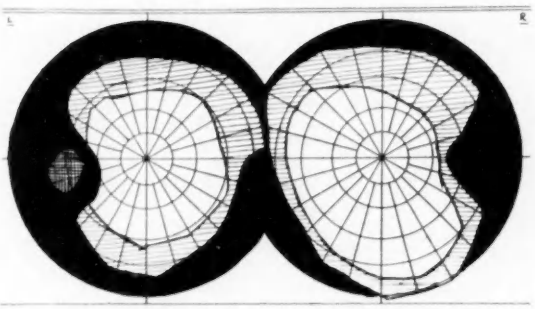
Case 2—Mrs. C. F. F., A-5348. Chart VII—March 8, 1929, 11 a. m., bright, sunny; vision, R. 6/6—, L. 6/6—; tension, R. 4/5.5=22 mm., L. 5/5.5= 18 mm.; 1.5/1000 outer field, good light; inner field, dim light.



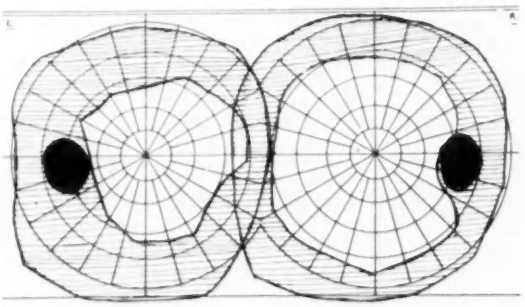
Case 2—Mrs. C. F. F. Chart VIIi—May 8, 1929, 12 m., bright, no sun; vision, 6/6— each; tension, 20 mm., each; 1.5/1000 outer field, good light; inner field, dim light.



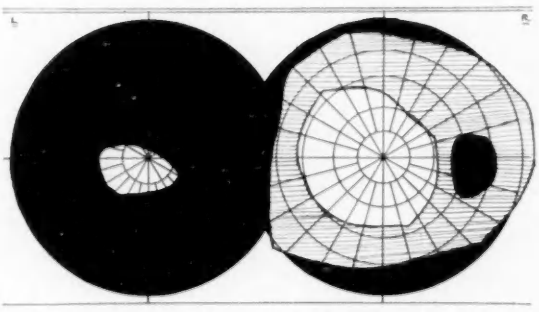
Case 2—Mrs. C. F. F., A-5348. Chart IX—Oct. 1, 1930, 11 a. m., bright, cloudy; vision, 6/6— each; tension, R. 22 mm.; L. 18 mm.; outer field and blind spot 1/1000, good light; inner field, 2/1000 dim light; 1/1000 not visible, good light.



Case 2—Mrs. C. F. F., A-5438. Chart X—Dec. 3, 1930, 12 m., dull, cloudy; vision, 6/6— each; tension, 18 mm. each; outer field 1/1000, good light; blind spot 4/1000, good light; inner field, 4/1000, dim light; 1/1000, 2/1000, not seen, dim light.



Case 2—Mrs. C. F. F., A-5348. Chart XI—Dec. 17, 1930, 10:30 a. m., bright, sunny; vision, 6/6 each; tension, R. 18 mm., L. 20 mm; outer field and blind spot 1/1000, good light; inner field, 3/1000, dim light; 1/1000 and 2/1000 not seen, dim light.



Case 3—Mrs. M. N. C., A-7599. Chart XII—May 10, 1930, noon, bright, sunny; vision, 6/6— each; tension, R. 30 mm., L. 35 mm.; good light, 1.5/1000, 12/1000; outer field, good light; blind spot 1.5/1000, good light; inner field, dim light.

to have occurred. However, the 2/1000 field is quite contracted with a reduced illumination and 1/1000 invisible. No explanation is offered for the increased size of the 1/1000 field in good light. In Chart X still further progress is noted, for at this time the 2/1000 field has become lost. Chart XI indicates a second apparent improvement, the 1/1000 and 2/1000 field remaining lost, however. These ups and downs corresponded fairly closely with the clinical course of the disease. The patient suffered recurrent attacks of pain, which were coming at more frequent intervals, although the tension was always found within normal limits. The depressions in her field were found when they were examined relatively soon after discomfort, the larger fields at a longer interval after the occurrence of subjective symptoms.

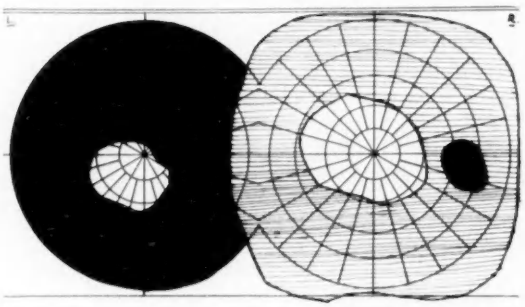
Case 3 has been under observation less than one year. Charts XII and XIII show improvement under treatment if the extent of the field and the size of the blind spot alone are considered. With the light reduced this improvement diasppears, the second field showing some reduction in size from the first.

In case 4 Charts XVI and XVII show some slight improvement in the field after operation. This patient has been under observation for nearly four years. An iridectomy was done on his right eye soon after he came under observation. Charts XIV, XV and XVI show the failure of his left eye, which was found more and more frequently to have elevated tension not fully controlled with miotics. It is interesting to note that the defects found in bright light are reduplicated and intensified in dull light.

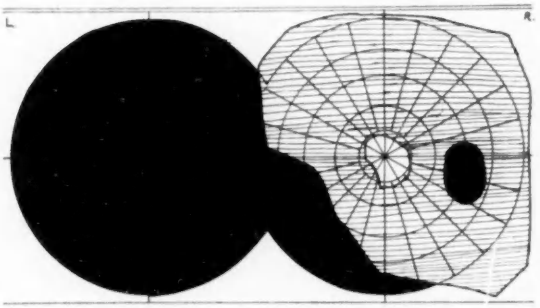
The remaining cases have been observed for varying periods, but only one field examination under the conditions stated is available, either because the patient has not returned for further observation or because the case is too recent.

Case 5 was under observation six months only and has not been seen since operation was advised. Chart XVIII shows the only changes to be enlargement of the blind spots and slight defects above, with full illumination, whereas a contraction more in accordance with the progress of the disease was demonstrable when the light was reduced.

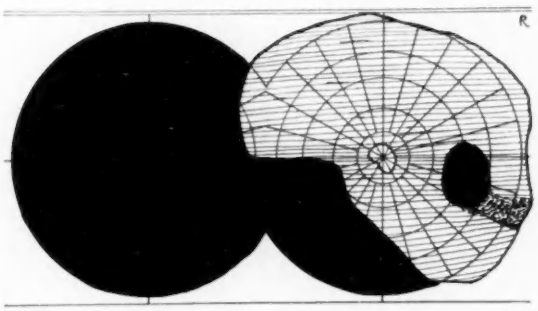
Case 6 has been under observation seven years. The left eye failed in spite of treatment with miotics, operation being refused. Under miotics the disease has appeared to be controlled, although



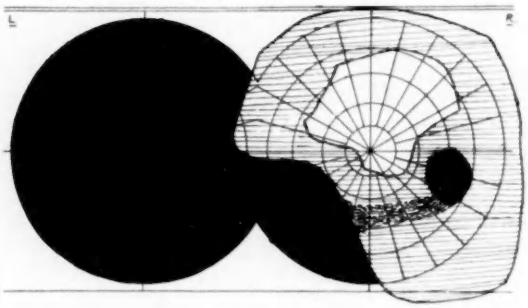
Case 3—Mrs. M. N. C., A-7599. Chart XIII—Jan. 2, 1931, 10:30 a. m., bright, sunny; vision, 6/6—; tension, R. 3/5.5=31 mm., L. 2/7.5=41 mm. (no drops for 12 hours); good light, 1/1000, 8/1000, 20/1000, at same points; 1/1000 test, outer field and blind spot, good light; inner field, dim light.



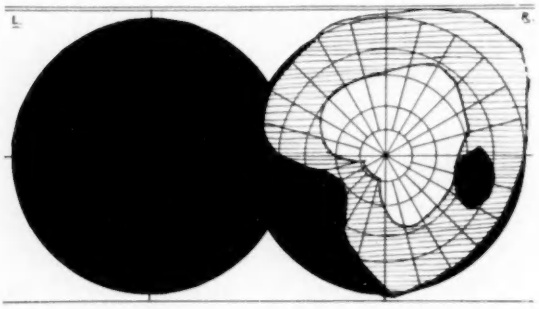
Case 4—Mr. I. H., A-6538. Chart XIV—March 17, 1930, 9:30 a. m., bright, sunny; vision, 6/6; tension, 4/5.5=23 mm.; 1.5/1000 test; outer field and blind spot, good light; inner field, dim light.



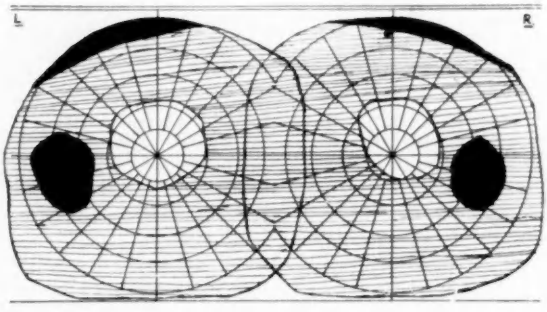
Case 4—Mr. I. H., A-6538. Chart XV—May 29, 1930, bright, cloudy; vision, 6/8—; tension, 6/7.5= 23 mm.; outer field and blind spot, good light; inner field, dim light, 1.5/1000 test.



Case 4—Mr. I. H., A-6538. Chart XVI—Sept. 30, 1930, 9:30 a. m., dull; vision, 6/8+; tension, 3/7.5=34 mm.; outer field and blind spot 1/1000, good light; inner field 2/1000, dim light; 1/1000 not seen, dim light.



Case 4—Mr. I. H., A-6538. Chart XVII—March 7, 1931, 9:00 a. m., bright day; vision, 6/10; tension, 5/5.5=18 mm.; outer field, 1/1000, good light; blind spot, 2/1000, good light; inner field, 2/1000, dim light; 1/1000 not seen, dim light.



Case 5—Mrs. J. H. B., A-6603. Chart XVIII—June 4, 1929, 10:30 a. m., bright, sunny; vision, 6/6+ each; tension, 3/7.5=37 mm.; outer field and blind spot, 1.5/1000, good light; inner field, 1.5/1000 dim light.

there have been occasional elevations of tension. Chart XIX shows a marked loss of field when the light is reduced. This corresponds fairly well with the symptoms, for although vision remains within normal limits it has fallen from 6/5 to 6/6—. He complains at each periodic visit that he does not see as well as formerly.

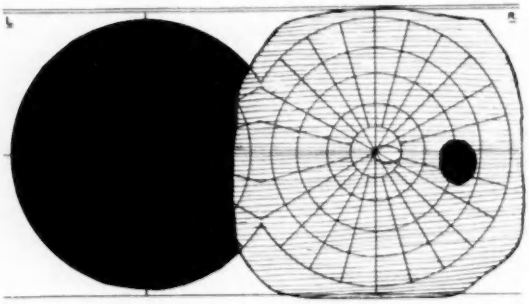
Case 7 came with a high tension but no pain. Miotics controlled the tension while he was under observation, a short time only.

Chart XXI of case 8 shows the connection of the scotoma found with full illumination with the peripheral field when the light is reduced and a larger test object is necessary. This patient was seen but once, for a second opinion, operation having already been advised by another ophthalmologist.

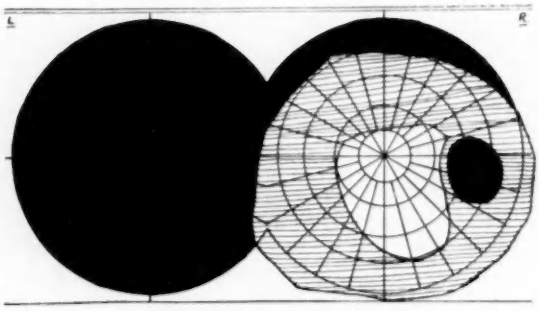
Cases 9 and 10, Charts XXII and XXIII, are clinic cases, both having been operated on since these observations were made. Case 9 was at an advanced stage, case 10 in an early stage, although one eye had already been lost.

Case 11, Chart XXIV, has been examined once. The more advanced stage of the disease in the right eye is more clearly evident in the field with reduced illumination. Although the tension was not found to be elevated at any time, both improved after the use of miotics, the improvement in the left eye being the greater.

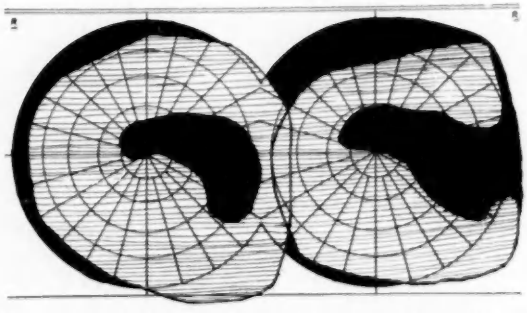
These observations are too few in number and extended over too short a period of time to allow conclusive deductions. While it is true that the procedure is crude and open to many criticisms, it is simple and may lead to earlier diagnosis than the usual method of scotometry. It should, however, be used with caution, only after the individual worker has determined for himself the limits of the normal field with the illumination reduced under his own particular working conditions or by making a comparison at the time of testing with a normal for control. In those cases in which repeated examinations have been made, the results appear to correspond closely to other clinical features of the case under observation.



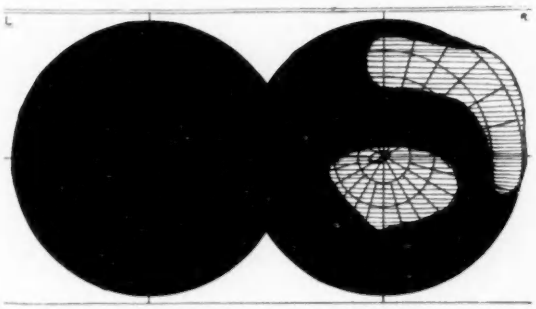
Case 6—Mr. G. C. B., A-2322. Chart XIX—Aug. 1, 1930, 11:30 a. m.; vision, 6/6—; tension, N. to fingers; outer field and blind spot, 1.5/1000 good light; inner field, 1.5/1000 dim light.



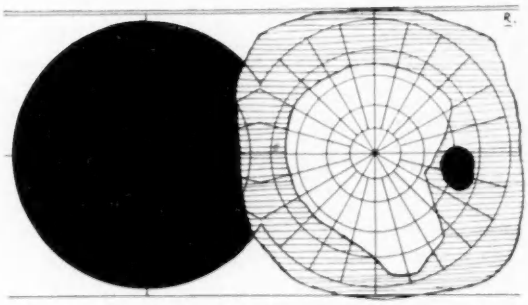
Case 7—Mr. W. B. C., A-7659. Chart XX—June 10, 1930, 10:30 a. m., dull day; vision, R., 6/12+; tension, R. 2.5/10=58 mm., L. 1/10=71 mm.; outer field and blind spot, 1.5/1000, good light; inner field, 1.5/1000, dim light.



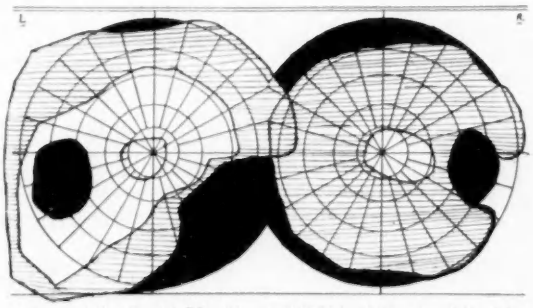
Case 8—Mr. M. G., A-7676. Chart XXI—June 19, 1930, 11:30 a. m., bright, sunny; vision, 6/12+; tension, 6/7.5=22 mm.; left hand field, 1.5/1000, good light; right hand field, 5/1000, dim light; 1.5/1000 and 2/1000 not seen, dim light.



Case 9—Mr. P. V., clinic case. Chart XXII. Vision, R. 6/10; tension, R. 32 mm., L. 34 mm.; outer field, 1.5/1000, good light; inner field, 3/1000, dim light; 1.5/1000 not seen, dim light.



Case 10—Mr. P. M., clinic case. Chart XXIII—June 2, 1930; vision, R., 6/10; tension, 3/7.5; outer field, 1.5/1000, good light; inner field, 1.5/1000, dim light.



Case 11—Miss M. L. S., 68-77. Chart XXIV—Feb. 4, 1931, 11 a. m., dull, cloudy, snowing; vision, R. 6/18—, L. 6/18+; tension, 25 mm. each.

Outer field, 1/1000, good light	Vision, R. 6/18— L. 6/18+	Outer field, 1/1000, good light
Blind spot, 1/1000, good light	Tension, 25 mm. each	Blind spot, 3/1000, good light
Inner field, 1/1000, dim light		Inner field, 2/1000, dim light
Middle field, 2/1000, dim light		1/1000 not seen, dim light

DISCUSSION.

Dr. Luther C. Peter, Philadelphia: Any method or practice which will increase the accuracy of perimetric technique is worthy of serious consideration. In glaucoma, as Dr. Marlow has stated, refinement of technique will uncover early evidence of inroad upon the visual field and such evidence is vital. The same, however, applies to all phases of visual field disturbances. By eliminating the variable factors the evidence becomes trustworthy and of diagnostic and prognostic value. It enables one to determine, with accuracy, whether treatment is accomplishing that which we hope to accomplish. Central visual acuity continues to be good in most cases until the disease is well advanced, and intraocular tension to all appearances, at least when the tension is taken, may seem to be satisfactory; but after all, the real measure of progress can only be determined by minute study of the fields.

The atrophy which develops in glaucoma differs from all other forms of atrophy. It follows logical and definite lines. Consensual contraction, as shown by Dr. Marlow's studies and method, furnishes valuable information. The same field defects, however, can be developed in simple atrophy if the same methods of study are followed. In this respect the same principle applies to the use of the reflecting ophthalmoscope when the source of illumination is furnished by gas instead of electricity, and the gas illumination can be raised and lowered at the operator's will. A nervehead which, to all appearances, is good in ordinary illumination, readily discloses its pallor in incipient cases when the illumination is reduced to a minimum.

The same method of controlled illumination as practiced by the essayist can be carried to a greater degree of efficiency by suitable modifications.

In looking over the charts of the cases which the author has reported I find central visual acuity is subnormal in all, ranging from 6/6 to 6/10 or less. If light sense were to be tested in each of these cases one probably would find it was definitely lowered in each instance. Contraction of the field, therefore, in subdued light would be expected, as is well shown in the cases reported.

The contraction is early evidence of definite pathology which is the result of increased intraocular tension.

The method, however, can be of even greater value in glaucoma in uncovering areas which naturally follow certain lines of development. In other words, one can not only uncover general evidence of lowered sensitivity by subduing the illumination, which is evidence of value; but to this can be added the strictly localized areas of physiologic block in conductivity and the evidence of localized atrophy which must necessarily follow such physiologic block if the intraocular tension is not relieved.

In order to uncover these areas the technique suggested needs some amplification. In my experience the Ferree-Rand slate or the Lloyd slate offer greater precision than examinations at one and two meters distance employed on the usual Bjerrum tangent screens. Illumination is under better control, fixation is more accurate, and the area of defect uncovered is more easily reproduced.

As a second modification, the use of colored test objects of proper size, especially green, as well as small white test objects, offers greater accuracy and efficiency in uncovering such defects than the simple use of small white test objects.

The central field studies thrown upon the screen will illustrate this. The right blind spot shows no enlargement for a minute test object for white, even in subdued light. The green stimulus, however, uncovers a Sidel sign of considerable magnitude in the same degree of illumination. The patient was quick to recognize this for herself.

The third modification which offers greater precision is that of following the course of the nerve bundles, which are so familiar to all of you. If the test objects are carefully passed back and forth along these paths in which, experience teaches us, the earliest evidence of invasion will be found in average cases, reduced illumination will often uncover areas of greatest intensity of early loss of function.

Ferree and Rand, in a recent paper, were able to detect such areas by means of reduced illumination with a blue test object. I personally find red or green test objects of greater value in this sensitive test.

In offering these suggestions or modifications of the methods used by Dr. Marlow I simply wish to point out how reduced illumination can add to the value of the principle which Dr. Marlow has so well presented.

Dr. Mark J. Schoenberg, New York City: It takes a certain amount of temerity to discuss Dr. Marlow's paper after Dr. Peter's exhaustive discussion. However, I think there are a few things to be added.

If you keep a patient with glaucoma in a dark room for half or three-quarters of an hour, his intraocular pressure will go up; an early sign of glaucoma. Dr. Marlow's method of comparing field of vision taken in reduced illumination with that taken in good light, brings out defects otherwise not discoverable. Why this defect becomes manifest is a question that is not easily answered. The defect can not be due to a sudden increase of intraocular pressure, because the hypertension takes place only after half to three-quarters of an hour of exposure to darkness. Since the rods are the retinal units involved in the visual function of the retina in reduced illumination, we may be justified in assuming that in glaucoma—if Dr. Marlow's findings are definitely confirmed—the function of the rods is impaired. Dr. Marlow's findings are so important that it will be worth while going a little deeper into the study of this subject. Perhaps a careful study of a hundred normal individuals, as a control, also of other conditions than glaucoma, like disseminated chorioretinitis, vascular retinopathies, etc., will shed more light on this question.

Another interesting point to be worked out would be the pharmacological phase of the question. How do miotics, mydriatics and cycloplegics affect the field of vision in glaucomatous patients, in dim and in bright light? May I ask Dr. Marlow whether he has tried to use ordinary dark glasses on these patients instead of dimming the light?

Dr. S. B. Marlow, closing: I simply want to express my appreciation of Dr. Peter's courtesy in discussing my paper and of what he has added to it.

As to Dr. Schoenberg's questions, it is impossible to answer them all at this time. I have had no experience with the use of dark glasses. If they should be used the general illumination would still have to be under control. This question emphasizes further the necessity of controlling the illumination, or at least of recording its amount. This has been done in a general way for years by noting whether the day on which the test is made is sunny or cloudy. The varying conditions of ordinary window lighting probably account for some of the variations in the field from one observation to another, and from one office or clinic to another.

MALIGNANT MELANOTIC TUMORS OF THE EYE: A STUDY OF HISTOLOGIC TYPES IN 111 CASES.

GEORGE R. CALLENDER, M. D.,*

WASHINGTON, D. C.

During the past nine years there have been received from the members of the †American associations supporting the Division of Ophthalmology 1772 enucleated eyes. Of these, 111 were the site of malignant melanotic tumors. Follow-up reports have been sent yearly to the physicians contributing these eyes, and responses have been received from a large proportion. While the length of time these cases have been followed is still inadequate, certain trends are indicated. It is believed that the presentation of data now available will stimulate those who have already contributed to follow up their cases, and possibly to contribute others, and will result in additional contributions of melanotic tumors, either completed or still under observation. In this way the number of cases can be increased with greater rapidity, so that in a relatively few years a sufficient number will be accumulated to justify definite conclusions.

The microscopical study of these tumors of the eye revealed that there were several easily distinguished histological types. These types are also seen in malignant melanotic growths arising in other structures of the body. This study is based on the histological types which have been found in 111 cases, and deals with the age distribution, location, pigmentation and prognosis. The histological types are described and illustrated below.

SPINDLE CELL TYPE.

The spindle shaped cell is more frequently found than any other. It forms the entire mass of some tumors, and is always found in the mixed form. The spindle cell tumors are composed of sheets, whorls and other irregular arrangements of spindle

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shaped cells, with long, oval nuclei. The ends of the cells appear to terminate in fibers, so that the cells resemble fibroblasts, but these fibers do not stain with differential dyes like those of fibroblasts. The cells lie closely packed and there is no argentophile reticulum except that accompanying nutrient vessels. These spindle cell tumors can be separated into two divisions by the nuclear characteristics.

Spindle Cell Sub-type A.—In this sub-type the nucleus has a delicate, reticular structure, in which the nucleolar material is not well defined. These tumors are usually fairly heavily pigmented. In this series, this sub-type has been found only in the uvea, but there are two cases of melanomas of this type in the Army Medical Museum collection, primary in the central nervous system, which were of slow growth, and killed by reason of pressure on vital structures, but did not metastasize. (Figs. 1 and 2.)

Spindle Cell Sub-type B.—In this type the cell has a sharply defined, deeply stained, small, round nucleolus, usually situated near the center of the nucleus, in a rather coarse nuclear network. The tumors of this type of cell are usually lightly pigmented. Many of them on gross examination are white tumors, leucosarcomas, but evidence of pigment formation by the tumor has always been found after careful search. This type of spindle cell is the one usually found in the mixed form. It has been seen in both choroid and ciliary body. (Fig. 3.)

EPITHELIOID TYPE.

This type is composed of polygonal cells, usually of relatively large size, though there is considerable variation in both size and shape. The nucleus is large, round or somewhat oval, and the nucleolus is distinct, small, round and often there are two nucleolar masses. Often rather abundant argentophile reticulum is found about and between the cells, and there are usually a moderate number of adult fibroblasts scattered throughout the tumor in addition to those of the stroma. The origin of the reticulum cannot be stated with assurance, but it is probably developed from the stroma of the growth. This type varies greatly in the degree of pigmentation, but is usually rather deeply colored. It has been found in the uvea and as a primary tumor of the limbus area. This cell type is found in the mixed cell tumor. (Figs. 4 and 5.)

Fig. 1. Spindle cell subtype A. Accession No. 31097. ×333.

Fig. 2. Same as Fig. 1. ×667.

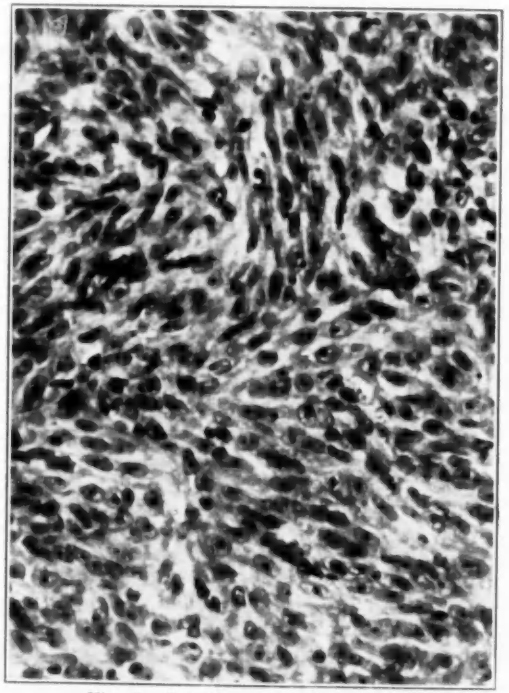

Fig. 3. Spindle cell subtype B. Accession No. 32790. ×333.

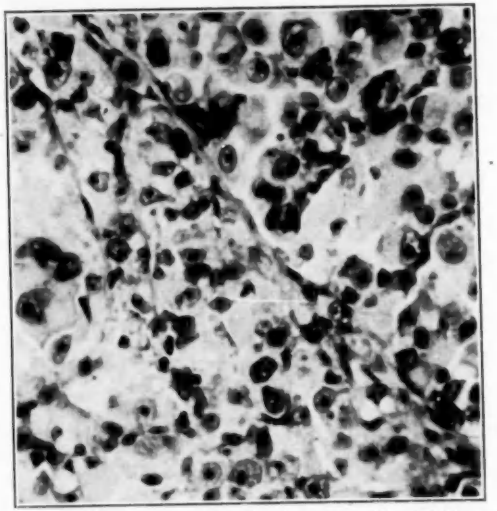

Fig. 4. Epithelioid type, large cell. Accession No. 24513. ×333.

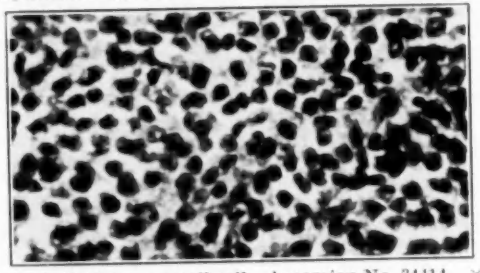

Fig. 5. Epithelioid type, small cell. Accession No. 34414. ×333.

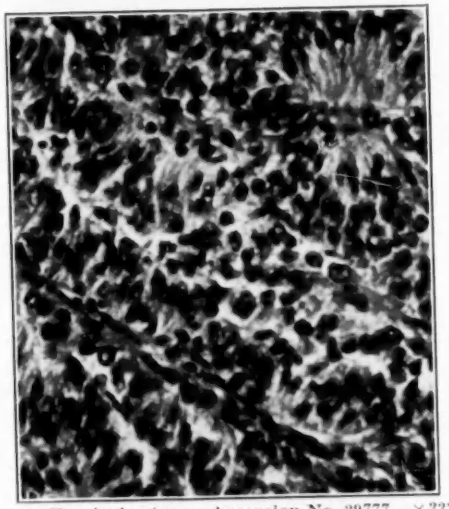

Fig. 6. Fascicular type. Accession No. 29777. ×333.

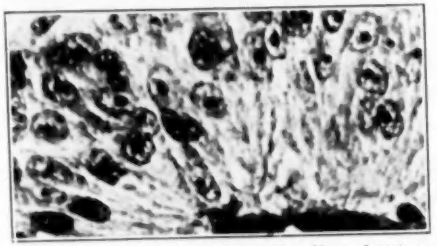

Fig. 7. Fascicular type showing palisading about vessel. Accession No. 29777. ×667.

THE FASCICULAR TYPE.

The cells of this type are elongated, sometimes forming fiber-like structures, but rounded and polygonal shapes are seen. The characteristic nucleus is oval, has a distinct nucleolus, and closely resembles the nucleus of the spindle cell sub-type B. The majority of the cells in this tumor are arranged in columns or fasciculi, the long axis of the cell being at right angles to that of the column. The cells radiate about the center of the column in a palisade arrangement, the center being a lymphatic or capillary blood vessel. In cross section of such columns there is often a pseudo-rosette appearance. Pigmentation is usually very scanty, and careful search of many sections may be required in order to demonstrate pigment production by the tumor. These tumors are often called leucosarcoma. This type is not peculiar to the eye, but occurs in the skin, either as a part of a mixed cell type or without other arrangement in the metastases from skin melanomas, the original tumor usually having a mixed cell composition. The fascicular arrangement sometimes is seen in areas of the mixed cell tumor of the eye. (Figs. 6 and 7.)

MIXED CELL TYPE.

This is the most common melanotic tumor of the eye. It consists of irregular mixtures of spindle and epithelioid types of cell, with occasional areas of the fascicular type. Sometimes the various types of cell are mixed in close association, but more frequently small areas are composed entirely of one or the other cell type. Sometimes the growth, which appears to be older, will be of spindle cells, with a smaller growth, which appears to be an extension, composed of the epithelioid cell. When two masses or lobules of tumor are found, these may vary greatly in the degree of pigmentation, the spindle cell portion usually being darker, though the reverse is occasionally true. These tumors are usually very heavily pigmented. They are occasionally found in the limbus area. (Figs. 8, 9, 10 and 11.)

From the histological standpoint, the epithelioid cells appear the most malignant, and mitotic figures are more abundant in them. The fascicular type appears next in order, and some of its cells often are of the epithelioid type. The spindle cell sub-type B appears more malignant than the spindle cell sub-type A, the latter being the least malignant appearing melanotic tumor. The

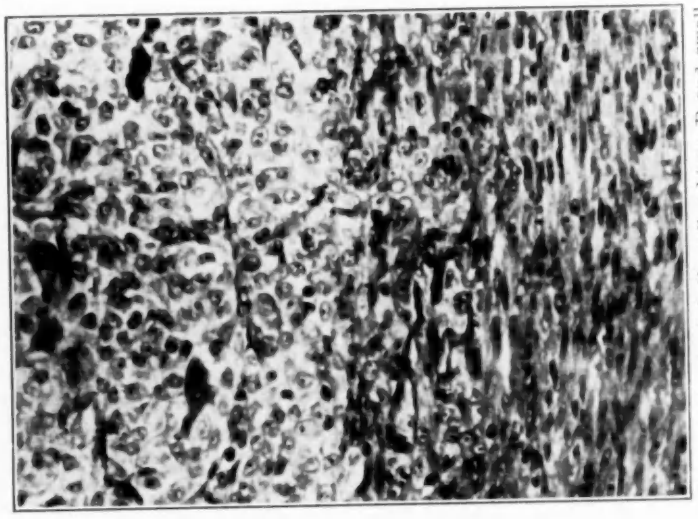

Fig. 9. Field A. Spindle cell subtype B and small epithelioid cells. Accession No. 2999. ×333.

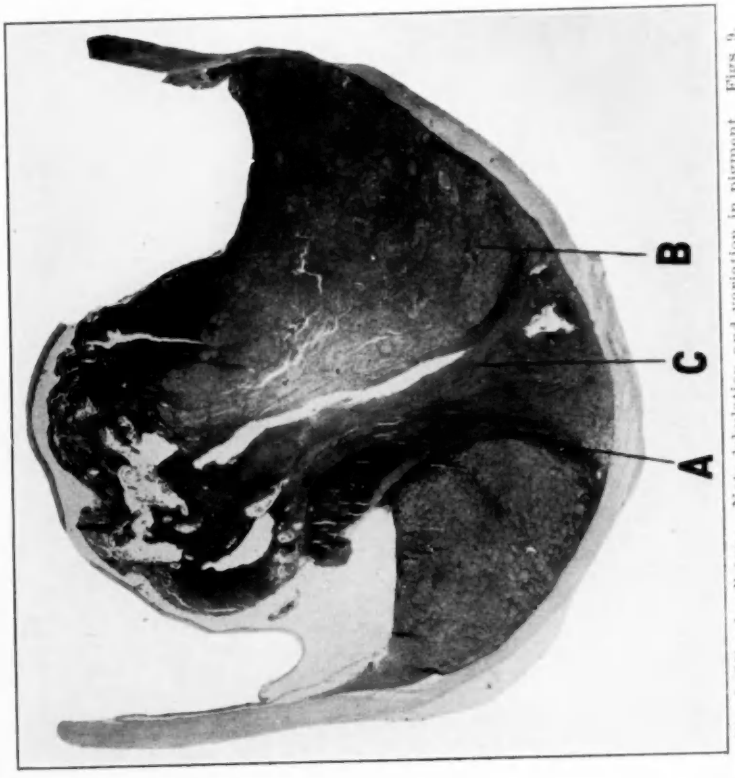

Fig. 8. Mixed cell type. Note lobulation and variation in pigment. Figs. 9, 10 and 11 are from fields marked A, B and C. Accession No. 2999. ×6.

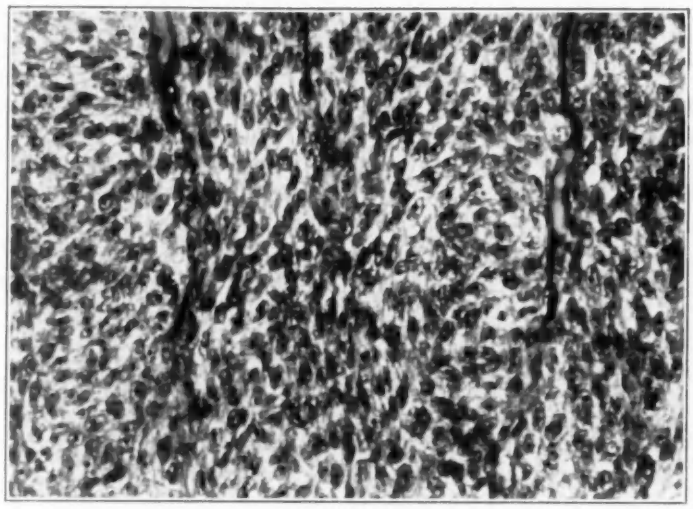

Fig. 11. Field C. Fascicular type. Arrangement not as typical as in Fig. 6. Accession No. 2969. ×333.

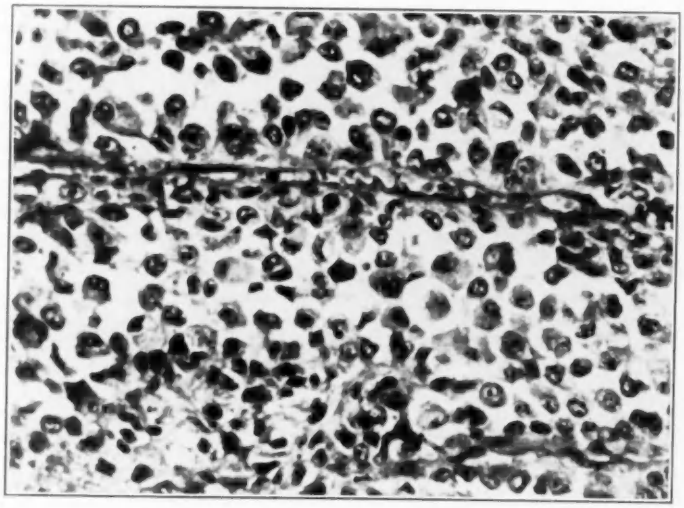

Fig. 10. Field B. Large epithelioid cells. Accession No. 2969. ×332.

mixed cell type varies greatly in its character by microscopical examination; in metastases the epithelioid cell usually predominates.

Spindle Cell Sub-type A.—Of the thirteen cases of this type, only one had an extension outside the globe. This one died from cardiorenal disease over five years after enucleation when over forty-five years of age. One case in the eighth decade died of pneumonia a short time following operation. Another case in the eighth decade died in the second year from unknown causes. Two cases were not followed at all, and another for only two years. Of the balance of eight, one is living over ten years after enucleation; one in the sixth year; one in the fourth year; one in the third year, and four in the first and second years. None of the thirteen cases is definitely known to have died from the neoplasm. The tumor in one case arose in the ciliary body; in the balance, twelve cases, it arose in the choroid.

Spindle Cell Sub-type B.—Of the twenty-two cases in this group there are no deaths reported from tumor. One died at age fifty-five of myocarditis; one in the eighth decade is said to have died from old age; two were not traceable, and one was lost track of after the second year. The balance, seventeen cases, are living; two in the sixth year; one in the fifth year; four in the fourth year; three in the third year; and eight in the first and second years after enucleation. Three of these cases had orbital extensions. One tumor arose in the iris; one in the ciliary body; and the balance in the choroid. Only two of the twenty-two tumors had sufficient melanin to be considered abundantly pigmented.

Epithelioid Type.—Of the ten cases in this group, six were in the choroid; one in the ciliary body; and three arose in the tissues at the limbus outside the cornea and sclera. One tumor arising in the choroid had extrabulbar extension. Three died of tumor, one in the third year, two in the second year. One died in the fourth year of tuberculosis, and one died in the eleventh year from unknown causes in the seventh decade of life. One of the cases in which the tumor was the cause of death had the original growth start in the limbus. Five cases are living: one over ten years; one in the sixth year; one in the fourth year; and two in the first year.

Fascicular Type.—There are but six cases of this type, all located in the choroid. With one exception, these cases had a

TABLE I.

CLASSIFICATION BY HISTOLOGIC TYPES OF 111 PRIMARY MELANOTIC TUMORS OF THE EYE, SHOWING AGE DISTRIBUTION, LOCATION, DEGREE OF PIGMENTATION AND OUTCOME AFTER ENUCLEATION.

	AGE AT OPERATION							LOCATION			PIGMENT			STATUS AFTER OPERATION												DEATHS		
	TOTAL	20 to 39	40 to 49	50 to 59	60 to 69	70 and over	Not known	Uvea	Limbus	Orbital extension from uvea	Slight	Moderate	Abundant	2 year or less		2 to 3 years		3 to 4 years		4 to 5 years		5 to 10 years		10 years and over		Patient lost track of	Tumor	Other	Unknown
														*	†	*	†	*	†	*	†	*	†	*	†				
SPINDLE Sub-type A	13		4	4	3			13		1	3	2	8	4	1	1	1	1				1	1	1		2		2	1
SPINDLE Sub-type B	22	3	4	4	8	2	1	22		3	11	9	2	8		3		4		1		2		2		2		2	
Epithelioid	10	2	3	2		1	2	7	3	1		6	4	2	2		1	1	1			1		1	1		3	1	1
Fascicular	6	2	1	2	1			6			5	1		1	2	2						1					2		
Mixed	60	9	10	17	13	9	2	59	1	11	3	15	42	15	9	11	7	3	2	4		3	1			5	15	3	1
TOTAL	111	16	22	29	25	14	5	107	4	16	22	33	56	30	14	17	9	9	3	5		8	4	2	1	9	20	8	3

*—Living. †—Dead.

pale or white tumor, requiring careful search to find any melanin formed by the tumor. Two have died of tumor: one in the first and one in the second year after enucleation. One is living in the eighth year; two in the third year; and one in the first year after enucleation. None had extrabulbar nodules.

Mixed Type.—In this group of sixty cases, no follow-up reports were obtainable in five. One case in the eighth decade died of unknown cause. Three died of other causes; two in the eighth decade, one from pneumonia two years postoperative, and the other from heart disease, two years postoperative; one in the sixth decade died a few months after enucleation following an appendectomy. Fifteen died as a result of the tumor.

Of the sixty cases, eleven had extensions outside the globe; four of these died of metastases, one in less than a year, one in the second year, one in the third year, and one in the fourth year. One died in the eighth decade, cause unknown. Six are living; two, in the fourth year after enucleation, two in the third year, one in the second year, and one less than one year since operation. The cases which had no apparent extensions outside the globe number forty-three. Eleven died of metastasis as follows: one in the sixth year, one in the fourth year, four in the third year, two in the second year, and three in the first year after enucleation. There are no reports on two. Thirty are living: one in the ninth year, two in the seventh, four in the fifth, one in the fourth, nine in the third, four in the second, and nine in the first postoperative year. One case was located in the limbus, a recent operation; four were in the ciliary body, two of which are in their fifth postoperative year, the other two in their first. One arose in the iris and is in the second postoperative year.

SUMMARY.

One hundred and eleven cases of malignant melanotic tumors of the eye have been separated according to histological type into five groups.

The results so far obtained suggest that patients with melanotic tumors of the eye consisting of spindle cells unmixed with other types have a more favorable outlook than those with epithelioid, fascicular or mixed cell types.

As no precedent was found for this method of classification of these tumors in a search through the Index Catalogue of the Army Medical Library, no bibliography is appended.

DISCUSSION.

Dr. Jonas S. Friedenwald, Baltimore: We are all deeply indebted to Major Callender not only for his beautifully clear presentation of this important study, but also for the interest and tireless diligence which he has for so long given to the ophthalmic collections of the Army Medical Museum. Our society has a very special interest in this work. The Section of Ophthalmology of the American Medical Association and the American Ophthalmological Society joined with us in 1923 to establish a special ophthalmic division in this national Registry of Pathology, thus enabling physicians all over the country to send specimens and get well-considered pathological diagnoses. But the contributions of the other societies have been mainly spiritual, while that of this Academy has been the material essence, without which the division would not have functioned. It is very fitting that the first important statistical study to emanate from this collection should be presented before this society.

Major Callender has analyzed his material so carefully that he has left me little room for comment. He has developed a most satisfactory system of classification for these tumors, and it is about that classification that I would like to say a few words. The neophyte might well ask, "What is all this classification about?" "What difference do these details make when all these tumors are supposed to come from the same general ancestry, and when in one and the same tumor various different forms may be found in close juxtaposition?"

I think, on the contrary, that the classification is significant and I shall try to show why. The early attempts to classify tumors based their distinctions on relatively gross morphological aspects. Later the attempt was made to identify the cell type from which the tumor originated. This represented a great advance, but it was soon discovered that tumors arising from apparently identical cells might take on very different appearances. All of the tumors that Major Callender has presented, for instance, arise so far as we can make out, from the same cell type. A great impetus was given the further study of those problems by the recent work on the classification of tumors of the brain, particularly that of Bailey. The adult, fully developed neuron, has numerous branching cell processes. In some types of brain tumors these mature cell forms are achieved. In others only blunt protoplasmic processes are present without fine fibrils, and finally at the other extreme we have cells which do not wait to mature at all before they divide and multiply. One can, therefore, arrange those cells in a scale according to the maturity of their development. In any given case wide variations may be found in different parts of the tumor, but usually a certain degree of maturity characterizes the majority of the cells. And this is of great clinical importance for it makes a great deal of difference in the rate of growth of the tumor as to whether or not the cells take time to mature before dividing.

The same sort of classification can be worked out in regard to these choroidal tumors. I have outlined such a scheme of classification elsewhere, and shall here only show that it is equivalent to the system that Major Callender has adopted. The adult pigment containing cell of the

choroid, the melanophore, is a large cell with branching protoplasms packed with pigment granules. If one follows this cell back into its embryological history one finds that it is derived from primitive mesenchymal cells resembling fibroblasts. Among the tumors of the choroid we occasionally find some made up mainly of mature melanophores. In following the cell types back from this mature form to the more primitive undifferentiated types we can watch either the decreasing complexity of the cell form, or the decreasing amount of pigment. The two do not always go hand in hand. We may have very primitive cell forms with a lot of pigment, or mature cell forms with little pigment. In any given case the degree of maturity of the cell forms is more constant than the amount of pigment. Major Callender has, therefore, chosen wisely in basing his classifications more on cell form than on pigment content.

The mature melanophore has long branching processes. More primitive cells are roughly cylindrical in shape with only blunt, stubby protoplasmic processes on their ends. When packed together these tend to arrange themselves side by side and constitute the fascicular type of Major Callender. More primitive cells still are spindle shape, while the least developed are round cells. What we want to know is the relative malignancy of these different tumor types. We cannot assume merely that the more primitive cell type is the most malignant. Perhaps a certain degree of maturity in the tumor cell is necessary to help it to survive in competition with the normal cells which surround it. This cannot be settled *a priori*. Only such careful studies as those of Major Callender will help us to disentangle this puzzle. We are indebted to him for having shown the way, and we should help him by sending him more of our specimens and by cooperating with him in filling out the clinical statistics.

Dr. Martin Cohen, New York City: I would like to ask Major Callender whether he conducted general autopsies in these cases in intraocular tumor? Also whether he tried to correlate the morphological tumor findings in the eye with those in other organs of the body?

Recently a case was brought to my attention with an iridocyclitis of long standing. This man did not wish his eye removed and insisted on evisceration of the eyeball. Four months later he developed a growth of orbital tissue which was very hard. He developed general symptoms. Eight months afterward he presented liver involvement and also signs in the lungs. I believe cases of iridocyclitis in which there is no visibility of the interior of the eye should not be subjected to evisceration because not infrequently in these cases of iridocyclitis melanosarcomata have been found. A safer procedure is enucleation.

Major G. R. Callender, closing: I wish to thank Dr. Friedenwald for his discussion, which has so materially added to the value of the paper.

Answering Dr. Cohen's question, very few cases in this series had autopsies performed upon them. The metastases from melanotic tumors usually show an embryonic type of cell, whether the original tumor was of this type, or less malignant appearing histologically. The correlation of the tumor in the eye with the metastases is of considerable importance, but unfortunately autopsies have rarely been done in the cases of the series reported here today.

CATARACT IN THE EYES OF FRESH WATER FISHES, DUE TO THE INVASION OF THE LARVÆ OF TREMATOID WORMS.

PARK LEWIS, M. D.,

BUFFALO, N. Y.

The presence of the larvæ of trematode worms in the eyes of many of our fresh water fishes is a fact that has long been known and has been frequently demonstrated. There are reasons, however, especially in connection with our later knowledge of the toxic effect of the secretions of parasites that take this subject out of the region of scientific curiosities or zoological research and make it at once important and opportune to the ophthalmologist. The simpler structure of the lower forms of life are so much more easily studied, the material is so much more readily available and the diseases from which they suffer are in many instances so closely analogous to those found in the higher vertebrates that the anatomical and pathological reactions in the cell tissues of the invertebrates may have an important significance in our study of the higher forms of life. The most obscure and malignant diseases are transmitted by gnats, flies, fleas and other insects, as well as by bacteria, so we are impelled to study the carriers and the way in which transmission occurs.

The opportunity, therefore, of studying the very interesting phenomenon found in a large number of fishes taken from the state hatcheries and placed in the aquaria of the Buffalo Society of Natural Sciences brought this subject again to our notice. In March last, Dr. Charles J. Fish, director, asked me to examine some fish that had recently been brought to the museum. In a number of trout in the aquaria, nine in ten were found to have opacities in the eyes of varying degrees of density. The fish were of all ages and sizes from the very newly hatched to those which must have been five or six years old. On examining the eyes of these fish with transmitted light, both with the ophthalmoscope and through a magnifying lens and later with the slit lamp, it was found that they all had varying degrees of opacity of the crystalline lens; mostly they were found to have clear corneæ.

After killing the fish and opening the lens, in the posterior portion of the cortex and under the capsule were found numbers of small objects in motion. The motion, as far as could be discovered under a loupe was undulatory, like that of an amoeba, but when the lens substance was teased out and the small moving object placed under a binocular microscope with an amplification of one hundred diameters, the details of an ovoid, nearly transparent organism, having a simple but most interesting internal structure, were perfectly apparent. It was subsequently determined that this was the larva of a worm of the family of trematodes, and the presence of these organisms in the eyes of our fresh water fish was so common as to be almost universal. It seemed, therefore, that it might not be without interest to briefly sketch the history of the discovery of these parasites in fishes' eyes, for it is not new, and the life cycle through which they pass, together with some of the implications to which their presence gives warrant.

In 1832, Prof. A. von Nordmann, himself a distinguished ophthalmologist, published in the "Microscopische Beiträge fur Naturgeschicte der Wirbellosen Thiere," a report of the astonishing fact to which we have just referred. He had found in the lens of many varieties of fish, trematode larvæ which he was able to classify as diplostomum volvens, the larva of the family of strigeidæ. "He counted," quoted Prof. LaRue of the University of Michigan, in his monograph on the "Strigeidæ," in which he summarized the observations of Butler and Berkhout, "in the lens of one fish (cyprinus erythrophthalmus) 270 larvæ and in the vitreous humor of the same eye 98; the total number was about 400."

Steentrup (1842) some ten years later and supplementing von Nordmann's observations by his own on the perch and the pike, says that von Nordmann's discovery of the entozoa, and especially of the trematode larvæ in the eyes of fresh water fish in such numbers that their presence in that organ might almost be considered as normal, caused at first, as might be expected, great astonishment, "but the fact is now generally recognized (in 1842) and as readily proved," and it was indeed looked upon as of such common occurrence at that time as to be hardly worthy of further comment, and it appeared to have been almost forgotten. It was occasionally referred to during the passing of the years by biological workers, but it interested ophthalmologists so little that it received but scant attention.

The publication of von Nordmann's was in 1832; the studies upon which this was based were evidently made in the preceding year, 1831. It is singularly appropriate, therefore, that in this centennial year of such an important ophthalmological discovery we should honor the memory of our distinguished colleague by recalling his valued contribution and drawing such conclusions from it as our more recent studies in immunology may warrant.

Neglected for nearly a hundred years, within the last two decades the study of these larvæ has awakened a new interest on the part of the biologists, and the entozoa in the eye of fresh water fishes has become a subject of intensive research. There is a very large number of representatives of the fluke family, some of those infesting the human kind, the liver fluke, the blood fluke (bilhartzia), the intestinal fluke and the lung fluke have been carefully studied and the life history is fairly well known. Barlow, who spent much time in studying the human intestinal fluke (the fasciolopsis buski) in China and the life history of which is now accurately known, says that 40 to 50 per cent of the patients of the Christian Hospital at Shaohsing, harbor this parasite and in some villages where routine examinations are made a hundred per cent of the people were found to be infested. It has been reported from India, Assam, Siam and the Malay Archipelago. It is a sub-tropical disease and is found in the greatest extent in China. In the Shaohsing area, which covers an extent of 1,600 square miles, the disease profoundly affects the lives of between a million and a million and a half of people. There is no evidence of these flukes ever reaching the human eye, but they may so affect the circulatory system that indirectly the eye may suffer.

Such an instance has been reported by Cecchetto in a recent number of the Annali di Ottalmologia e Clinica Oculistica. This is the first case of schistosoma mansoni (the bilhartzia), which has appeared in Europe with grave ocular complications. It is wholly a tropical disease, but travel from the Orient is so common and people from our own country are so constantly visiting in Europe that the possibility of acquiring this parasite is by no means remote. This case, indeed, appeared at the clinic at Verona, but the patient had made frequent trips to Venezuela, where the disease was common. There was no evidence of the worm having entered the eye. The sudden loss of vision was found to be due to an embolus of the inferior internal branch of

the central artery of the retina. These are, of course, wholly different from the fluke found in the eyes of fishes, but it is well to mention that they belong to the same family in our study of the fluke. While the entire life cycle of many of the fluke family has been discovered, in others some important links are still missing. In the study of these parasites in the eyes of fishes the observations of von Nordmann are now regarded as of basic importance, as the life cycles in all are very much alike.

The fluke is an animal of very low development and of the order of the flat worm or plathelminthes. They are parasitic during the larger portion of their lives. The name comes from the suckers with which they attach themselves (the word trematode meaning pierced with holes), and the special order as to whether there is one main opening or two, distinguishes the monostoma and the distoma. There are said to be about 2,000 varieties, some of which have been completely studied and others in which the life cycle is imperfectly known. The adult distoma volvens, which is very commonly found in the eyes of fishes, is barely visible to the naked eye. It attains a length of about one-tenth of a millimeter. The digenetic trematodes have a most complicated life cycle, living in different forms through different hosts. The adult is leaf-shaped or ovoid and at times nearly cylindrical. There is a well developed sucker around the oral opening and another secondary sucker called the acetabulum on the ventral surface. In most species the oral sucker is at the anterior end of the body but is not necessarily always so placed. Within the oral sucker is a pharynx or muscular sphincter which leads into an esophagus which bifurcates before the middle of the body, opening into two blind passages or ceca. The life history is most remarkable. The host of that generation producing eggs is ordinarily a vertebrate. The animal lives through two other forms of life in completing its cycle, in some instances three and even more. In the case of the strigeidæ, which are so commonly found in the eyes of fish, the vertebrate host usually is from predatory birds, either the herring gull, the duck or some other water bird by which the fish is devoured. The animal is parthogenetic as well as marital—that is, it is self-fertilizing, although the sperm cells may be carried from one to another. The fertilized egg in the body of the definite host must in order of its development reach the outer world. Most flukes live in the intestinal tract or adnexa of the host. The mature eggs are carried by the droppings

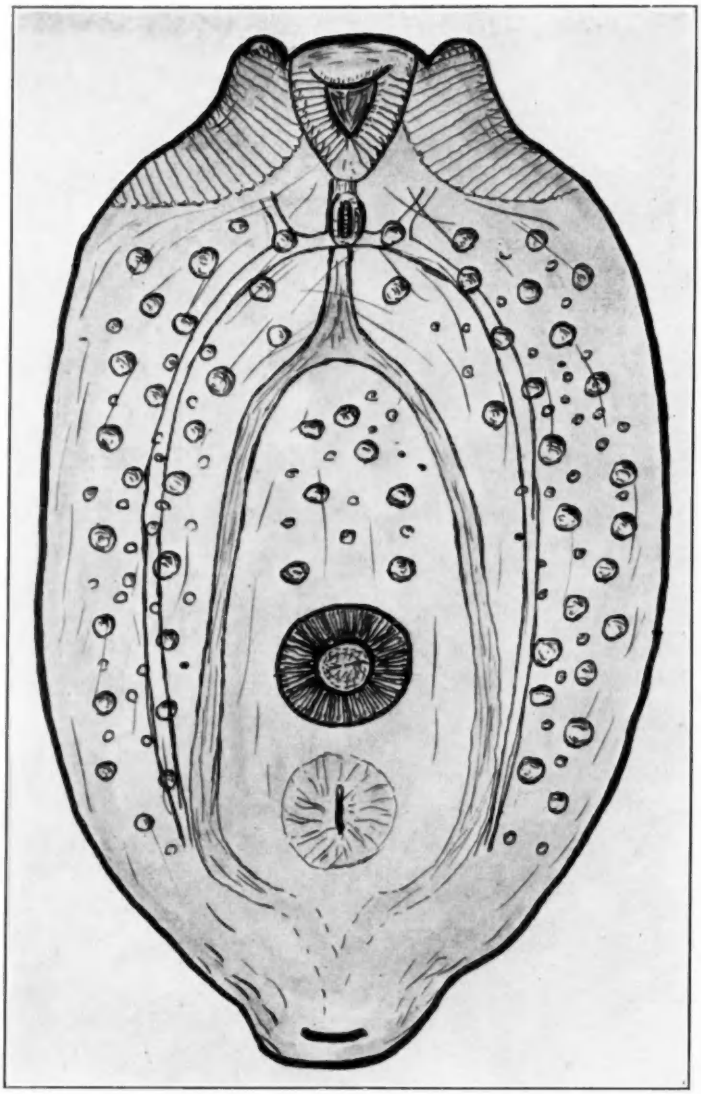

Fig. 1.

of the bird into the lake or pond which is isotonic or slightly hypotonic and in which the active embryo breaking through its shell coating becomes a free living organism. It swims by means of the ciliated epithelium and is termed the miracidium (literally little boy). Swimming actively, if it comes within the neighborhood of an appropriate species of mollusk, usually a snail, it swims directly to it and attempts to penetrate into the mollusk. Striking soft tissue it attaches itself by its glandular secretions and digests its way into the snail. Reaching a natural lymph channel it gradually migrates toward the apical end of the mollusk; it has in the meantime lost its swimming cilia and becomes encysted; it performs its digestive functions by osmosis. These sporocysts develop into larvæ, a new type known as redia. These when mature burst the cyst wall and migrate through the tissues of the snail. The rediæ are simple organisms with a sucker and an unbranched blind pouch for a digestive tract. Like the sporocysts these develop germinative cells within their bodies, and these develop into a second generation of rediæ, ultimately escaping from the body wall of the parent. In this way even a third generation may be produced. These escaping from the snail are entirely different from either the embryo or redia. These are called cercariæ and are furnished with a sucker, a forked digestive tract and an actively moving tail. The tail develops before they leave the snail. The number of cercariæ is enormous. A single fluke may produce as many as 50,000 eggs. These cercariæ swim about for a day or two. Coming in the vicinity of any prospective fish host they swim directly to it, puncture its sides and proceed to migrate toward the eye. The movement of the metacercariæ or larvæ as found in the lens is exceedingly interesting. The body appears to be enveloped in an elastic coat, by which it changes its form, rhythmically contracting from a circle into an elongated ovoid, constricted in the center and slowly coming back again to the circular form. These motions are continued with the regularity and rhythm of slow breathing. It is quite possible in those taken from the lens of the eye to see the slow extrusion from the oral opening of the ingested crystalline material. In one of the specimens observed by Shropshire and myself, a mass at least as long as one-tenth of the entire parasite had accumulated and could be seen as it was slowly discharged from the oral opening. The discharge from the oral opening had the same glistening appearance as that of the lens substance itself.

It is not our purpose to describe in detail the form of these parasites. This has been done very much more completely by LaRue and his students in the various monographs that have been issued from time to time during the past few years, and the general life history of the fluke has been given in detail by such modern parasitologists as Faust and Chandler. The extent of these infestations is very great.

In a report made to the Michigan Academy of Science, Miss Butler reported that 79 specimens belonging to seven species of fish were examined and parasites were found in 50 per cent of the trout perch, 60 per cent of the rock bass and 72 per cent of the yellow perch. All the parasites found in the eyes were in the

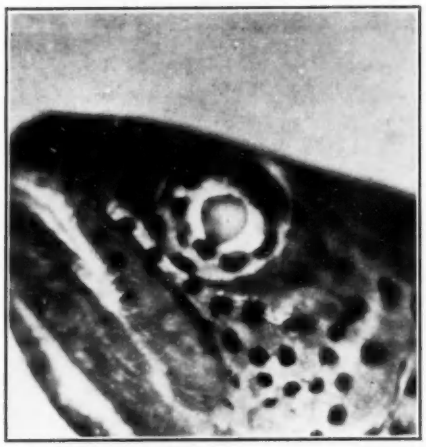

Fig. 2. Cataract in the eye of a fresh water fish.

free living state. They occurred within the pupil, in the anterior and posterior chambers, on the iris, in the pigmented tissue of the choroid lining, and most commonly in the vitreous humor. Their presence in such large numbers indicates that the eyes of these fish form a natural habitat for diplostumulum and tetracotyle.

The presence of these larvæ is not always harmless. Roth observed larval forms which he determined as diplostomum volvens in the eyes of chanchitas which produced a high grade exophthalmia. Later the lenses were completely clouded so that the fish were blinded and since they were able to take food only with difficulty they finally starved. Roth found these parasites between the lens and the lens capsule. In the report made from fish taken

from Douglas Lake, Michigan, during the summers of '18 and '19, some exceedingly important data were secured. In twelve varieties of the common fish which were either caught in nets or by hook and line, and others picked up or found dead on the shore, 349 were examined, and of these 249 were found to be infested with metacercariæ in the eyes. The total number of diplostomum found was 7,703, while with other varieties the total was 8,602 in this relatively small number of eyes. Some of the fish were very young, others over 32.1 cm. in length, indicating that they had reached their mature growth.

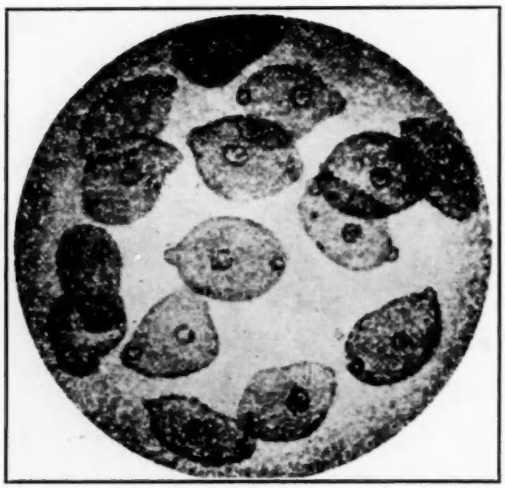

Fig. 3. Metacercariæ from the crystalline lens of the trout. (Plehn: Fischkrankheiten.)

One of the observers writes that the metacercariæ occurred practically always in the lens capsule of the sucker. Another reports that they are frequently found in the lens capsule and in the vitreous humor. A large number of these were the diplostomum volvens of von Nordmann. One of the most important observations was made by Szidat, writing in 1924. He frequently found living larvæ in the eyes of the stickleback, almost without exception in the lens, most commonly under the lens epithelium, where they are capable of movement. They occurred in large numbers. Thirty or forty in an eye was not uncommon. As these metacercariæ are almost transparent, outwardly the lenses of the eye show no visible changes except for a greater or less turbidity

of the lens, he could note nothing more of the presence of the worms. The worms are glass clear and transparent so that a severe injury to the ability to see does not appear to occur, at least, for example, sticklebacks, which proved later to be heavily infected, showed no difference in their behavior from their parasite-free fellows of the same species.

The most important observation was that of the penetration of the forked tailed cercariæ with pharynges into the fish. When

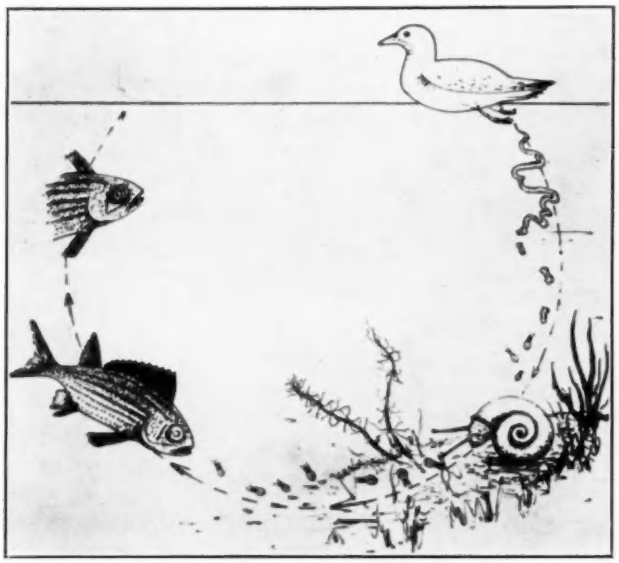

Fig. 4. Life Cycle of the Trematode Larvæ.
Eggs are laid in the gut of the water-bird; are hatched in the water; the miracidium enters the snail where eggs are laid producing the cercariæ. This finds an exit through the body of the snail into the water through which it swims, entering the body of the fish and shortly reaching the eye and penetrating the crystalline lens producing cataract.—Shropshire.

these penetrated in to great numbers they caused the death of the fish. They entered through all the surfaces of the body, but were prevented in places by the bony plates and scales. A microscopic examination showed them in all soft tissues of the body. Six days after penetration they could not be found in the brain, blood vessels and other organs but only in the eyes and chiefly in the lenses.

Dr. Ralph F. Shropshire, curator of the Buffalo Society of Natural Sciences, to whom I am indebted for invalued assistance

in the study of this problem and by whom all of the photographs were made, found flukes in the vitreous humor with the exception of one case; in all instances these flukes were found within the lens capsules. The largest number of flukes noted was found within the eyes of catostoma nigricans—a hog sucker. In this particular instance ninety-three of these flukes were found within the capsule of a single lens, and all flukes appeared to be in practically the same stage of development; and further, all of these flukes were extremely active when liberated from the lens.

During the course of a series of investigations of catostoma commersonii (common sucker), myxostoma breviceps (short-headed red horse), and catostoma nigricans (hog sucker), obtained from the waters of Cattaraugus Creek, within a few hundred yards of the new bridge under construction near Irving, practically all of the fish examined were found to be infested with these parasites. Anywhere from one to forty-six were to be found in a single lens and in addition marked corneal injuries were apparent. It might be worthy of note at this point that the commercial fishermen, men who for years have been taking fish from the creek and who may be considered as interested, although possibly not any too well trained observers, report that this year the majority of the fish which they have taken appear to be affected with a condition which they term "white eye." "My own examination of these fish," says Shropshire, "shows that nearly 90 per cent of them had cataract; that about 25 per cent, in addition to being cataractous, showed signs of a pathologic condition of the cornea which in most cases appeared to be traumatic. Further, it almost invariably followed that the greater the lenticular opacity the greater the corneal injury. In many cases in which the lenses contained parasites one could easily discern the track of the parasite within the lens. Usually this appeared as an irregular opacity extending from immediately within the capsule well into the nucleus and terminating with an actively moving fluke. Small vaccuoles were to be seen within this opaque track, and one may surmise that these vaccuoles contain products of the metabolism of the flukes."

An additional observation made on flukes removed from lenses by Shropshire was that these appeared to be trophic and that when mounted on a slide and under examination a marked increase in activity could be associated with the intensity of light to which the flukes were subjected. Miss Butler of the University of Michigan

has reported observing within the alimentary tracts of flukes, substances of a nature markedly resembling that of the crystalline lenses. We have observed on a number of occasions the excretion of substances from these flukes which might easily have been undigested portions of the lenticular tissue upon which they obviously appear to live. A careful study of the flukes found would indicate that they were identical with one of the strigidiæ described by one of Dr. LaRue's students at Michigan as a new species and named by them deplostomulum gigas.

There are a very few old records of flukes being found in human eyes. One was described by von Nordmann, in which eight specimens of monostomes were found in a cataract removed from the eye of an aged woman. The agamodistomum ophthalmobium was obtained from material from a congenital cataract in the eye of a child five months old. These were described by Geschiedt, who examined them microscopically. These cases occurred in a region where trematodes were abundant and have been actively discussed. The parasite was variously considered as having wandered in from the mother before the child was born or more likely was taken in with infested drinking water. A third case was that of Greef, who found two dead shrunken trematode larvæ in a unilateral cataract removed from the eye of a fisherman. Dr. Ward, professor of zoology in the University of Illinois, states that these cases are so rare as to be isolated instances and that no trematodes normally attack the bulbus oculi, but he says it is certain that other cases of similar character will be found from time to time in this country where none have yet been reported. When they are met it will be important for the observer to secure all data possible and to record the same with maximum accuracy, that some definite conclusions may be drawn, first as to the exact kind of parasite present and second as to the means by which it has reached this unusual location.

Japan, where raw fish is so commonly consumed and where trematodes are abundant, should some time bring forth such reports. The paragonimus or lung fluke found in the lung of the dog, hog and cat, has already been reported by Japanese observers to have ophthalmic involvements.

To the zoologist the life cycle of this parasite is a subject of great interest. The differentiation of the species and the small distinctions in structural formation greatly concern him. Very much attention is being given to these details, which are, indeed,

important from our viewpoint as ophthalmologists. There are also, however, certain fundamental facts which compel our attention.

First, that in the relatively vast spaces of the tiny ponds, in which the mudfish and suckers are found as well as in the greater area of the lakes and rivers which are the homes of the stickleback, the perch and the active trout, to say nothing of the enormous expanse of the great lakes in which are found the herring and the sturgeon, the microscopic free swimming cercariæ strike for their prey with such an amazing directness that their victims, in some instances like a Saint Sebastion, punctured with innumerable arrows, are destroyed by the onslaught. In others, where the infestation is not so great, after the first attack the fish appear to be unharmed, until in a comparatively short time practically all of these invaders have passed from the other organs into their ultimate goal and to that special structure in the eye which is apparently their definitive objective, the lens. This, of course, cannot be by chance. It has been explained as the effect of chemotaxis, which makes it almost more mysterious. What possible influence can pass from the fish to this minute living speck that draws it as though it were in a magnetic current, through what is to it relatively a world of space to the host that is to carry it on to its further development? It is true that the eggs that the fluke deposits are innumerable, but that fact in no way lessens the mystery of the influence that draws them towards their host. Even more remarkable is the force that takes them to the eye and more directly to the lens as a first resting place. Indeed, had the scheme been planned with the most exquisite finesse it could not have been more shrewdly chosen. Were one of the vital organs selected as its field of operations, such, for instance, as the heart, or the gills, or the liver, the fish would be killed and the source of supply destroyed. But the minute almost transparent flat embryos having gained access into the vitreous and through the capsule and into the lens enjoy a luxurious feeding ground for months and perhaps for years without more damage than to make turbid the rounded lens and lessen the value of sight, already one of the fishes' least important senses.

May not the fish, in this long struggle on the part of the lens against attack have developed a certain immunizing quality? May there not be chemical elements within the fishes' lens, within the fluke, or the metacercariæ, or possibly in the secretions elimi-

nated by the parasite that may have a protective value in developing resistance to one of the commonest causes of human blindness, cataract?

To sum up, we may surmise or assume that in many cases the lenticular opacity is caused by the actual mechanical irritation of the parasite within the capsule. However, it is our belief that in addition to the injuries caused by mechanical irritation there is, as a result of the metabolism of the flukes themselves, a substance excreted which in itself either acts directly upon the lens fibers or indirectly as an inhibitory factor of the normal nutritive process required to keep the lens fibers in their specialized state of transparency. Consequently a number of modes of attack of this problem present themselves. To us it would be extremely interesting were it possible by micro-chemical means to determine the nature of the excretory products of these flukes, and then naturally to ascertain the effect of the introduction within the body of another animal of this material. Is this toxin a specific—in other words, does this toxin (if it be a toxin) specifically act upon the lens tissues? Does this material which is excreted by the flukes cause any changes in the vitreous or the other fluids of the eye which might affect the nourishment of the lens; or does it cause any change in the permeability of the capsule of the lens which might likewise inhibit the proper nutrition of the lens? These are only a few of the numerous questions which obviously present themselves during the course of an investigation of this type, and are brought forth here primarily as suggestions for subjects for further research.

We may safely leave the details of the study of the life cycle of the trematodes to the entomologists, but as ophthalmologists we cannot fail to be deeply interested in a subject so intimately associated with our own studies on the physiology and pathology of the human eye.

The writer wishes to express his indebtedness to the work of Professor George E. LaRue and his students of the Department of Zoölogy of the University of Michigan, whose researches on the Strigiedæ embodied in a number of monographs were kindly placed at his disposal, and to Dr. Ralph E. Shropshire, Curator-at-Large of the Buffalo Society of Natural Sciences, without whose generous cooperation these studies would have been impossible.

DISCUSSION.

Dr. Harvey J. Howard, St. Louis: The parasites of the animal kingdom that directly affect human beings are roughly divided into three groups: (1) the protozoa, or unicellular organisms; (2) the helminths, or worms; (3) the arthropoda, or the insects and their allies. The helminths, or worms, are further divided into the flat worms and the round worms; and then again the flat worms are subdivided into the trematodes, or flukes, and the cestodes, or tapeworms. As ophthalmologists, we are more familiar with the tapeworms than we are with the flukes, since infections of the eye and orbit are occasionally reported as caused by *Echinococcus granulosus*, and *Taenia solium* and *Taenia saginata*, the latter two in their larval state being known as cysticerci. However, as one studies the literature he finds that trematodes have also been reported as involving the human eye. But strange to say, although the literature on the subject dates back to von Nordmann, one hundred years ago, only two cases have been reported in this century, and those about twenty-five years ago—by Greeff in 1905 and by Salzer in 1907.

I believe that Dr. Park Lewis' interesting paper on fluke infection of the eyes of fresh-water fish is not only valuable in reviving an interest in a phase of ophthalmology which has been practically forgotten the world over, but also that it bears implications which should claim the serious attention and study of all ophthalmologists. Dr. Lewis has brought up some interesting questions, such as the possibility of the fish having developed a certain immunizing ability against fluke infection, and that the lens of the fish may have a chemotactic effect in drawing the fluke to it. In studying this subject I have thought of another problem which, it seems to me, has even a more practical significance.

Before introducing this question I want briefly to make certain statements regarding the flukes that are known to infest human beings. There are intestinal flukes, blood flukes, liver flukes, and lung flukes, and flukes that apparently invade all parts of the body. All of these flukes have complicated life cycles. They all require a definitive or primary host, which is generally some vertebrate species. They also require an intermediate or secondary host in the form of a snail, and some flukes require a second intermediate host, such as a fish, or another mollusc, or even some form of plant.

A number of cases have been recorded of human beings in the lens of whose eyes were found larvæ of certain forms of flukes. Drinking water from ponds or lakes infested with this species of fluke apparently would not bring about an involvement of the lens by these larvæ, since the cercariæ, or the free-swimming larvæ of the kind of fluke (*distomata*) that has been found in the eye, cannot withstand the action of the gastric and intestinal secretions. Their larvæ are killed in a few minutes by these secretions. It is only the encysted larvæ of the intestinal fluke that can live in the stomach and intestines of man. In this instance the man acts as the definitive host of the fluke, which attains sexual maturity within his intestinal tract.

Recently there have been reported several cases of severe dermatitis in humans caused by the penetration of the skin by the larvæ of a certain

fluke. In his paper Dr. Lewis refers to the work of LaRue and others, who have identified various species of trematodes in the eyes of fishes. Szidat made the observation that forked-tailed cercariæ with pharynges will penetrate the skin of fresh-water fish, and after migrating through the body eventually lodge in the lens of the eye. Finally, we have the evidence that several human beings have been similarly affected, one of these cases being a fisherman with the larvæ of a fluke in both lenses. If, therefore, the larvæ of some flukes can penetrate the skin of a man, as evidenced by a severe dermatitis and the finding of the larvæ in microscopic sections, and if the larvæ of a known fluke, swimming about in fresh water, can penetrate into a fish, migrate through its body, and come to its final resting place—the lens of that fish—and if the eyes of humans may be similarly involved, is it not possible, and probable, that a human being who enters infested water may have his skin penetrated by such larvæ, and that these may migrate by way of the lymph and blood streams and finally reach the eyeball and the lens of the eye, just as they do in the fish? Possibly some of the reported cases of cataract caused by the larvæ of flukes were contracted in this way—i. e., through the skin while the individual was wading or swimming in a pool containing these larvæ. The implication is therefore quite plain, that it may be a dangerous thing for the sight of an individual to enter such an infested body of water. This is a new theory, I know, but I believe I am justified in proposing serious consideration of it in view of the composite picture that can be built up on the knowledge that we now have regarding flukes.

I want to take this opportunity to tell you briefly about a very interesting and unique case. I had in China a friend, a medical missionary, who was scientifically minded, and who became interested in parasitology, for he was living in an area where there are great numbers of flukes. He tried to work out the life cycle of an intestinal fluke known as *Fasciolopsis buski*. It was impossible to do so, however, because he did not have the proper human material for his experiments; ergo, he decided to be the experimental animal himself. After swallowing several dozen of the encysted larvæ of this fluke he found that it took about three months for the encysted flukes to become sexually mature in his intestinal tract. He infected himself a number of times in the same manner, and in the course of time suffered a great deal from gastro-intestinal symptoms, such as nausea, vomiting and abdominal pain. So far as he knows, he never had any fever.

I might add that shortly after swallowing his first dose of flukes he returned to America on furlough, and at certain laboratories in the East finally was able to work out the life cycle of *Fasciolopsis buski*, because he found it possible to provide himself with live specimens at a few hours' notice. A year or more later he was back again in China, and I met him at a medical meeting in Hongkong. He consulted me about one of his eyes which had been troubling him increasingly for a period of several weeks. I found that he had a severe episcleritis with a mild iritis and a moderate infiltration of the cornea, associated with some loss of visual acuity. He stated that he had been swallowing flukes over a period of about two years and was carrying an intestinal crop of them at that time. He

agreed with me that it was time to rid himself of the infection, which he did with large doses of carbon tetrachloride. Local treatment was also advised. I learned later that his ocular affection had cleared up.

I cannot definitely state that this case of inflammation of the anterior segment of one eye was due to the patient's infection with intestinal flukes, but I think the implication strongly favors it. In this instance, probably, the toxins associated with the infection, rather than the presence of larvæ themselves in the tissues affected, caused the ocular inflammation. As far as is known, the larvæ of intestinal flukes do not migrate through the body as do some of those distomate flukes which have been found in the eyes of fishes and of humans. In this matter, therefore, the case I am reporting here is not analogous to the infection of fishes which Dr. Park Lewis has described today in such a lucid and interesting manner.

THE HYALOID MEMBRANE OF THE VITREOUS.*

ALFRED COWAN, M. D., AND W. E. FRY, M. D.,

PHILADELPHIA.

In a paper presented by one of us (A. C.) before the American Ophthalmological Society, in June, 1931, a membrane was described that was seen by slit lamp examination after intracapsular lens extraction for cataract. This membrane was found in all uncomplicated cases examined, and it was seen at the earliest time after operation that slit lamp examination could be made. At first the membrane had a bulging anterior surface, but later it occupied a vertical plane just posterior to the iris and separated the aqueous from the vitreous chamber. At that time the opinion was advanced that this structure was not a newly formed structure, a postoperative formation or a condensation surface of the vitreous, but a membrane that existed in the normal eye.

The present contribution offers anatomical and histological evidence that such a membrane is present in both human and animal eyes. The work was done on pig, monkey, kangaroo and human eyes. Two types of preparation are presented, the first gross and the second microscopic. In the first type, which was carried out on pig, monkey and kangaroo eyes, after fixation in 10 per cent formalin, and further hardening in alcohol, the whole cornea was removed at the limbus and the iris cut off at its base. The anterior portion of the suspensory ligament of the lens was severed and the lens extracted in its capsule. Then the posterior portion of the sclera with the retina were cut away at a point about 1 mm. behind the position of the ora serrata. Next, under a dissecting microscope and high illumination, the vitreous was carefully removed. This left a membrane-like structure having its attachment in the pars plana of the ciliary body, which had completely separated the aqueous from the vitreous chamber.

In preparations of the second type, which were carried out on pig and human eyes, the cornea, iris and lens were removed as

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noted above. The specimen was examined by slit lamp, then dehydrated, embedded in celloidin and microscopic sections prepared. After this, sections were stained with the purpose of demonstrating a membrane which, from previous clinical examination and from the above gross anatomical preparations, we believed was present. The following stains were used: hematoxylin and eosin, Van Gieson, Mallory's analin blue, fuchsin, and phosphomolybdic-acid hematoxylin, Unna's orcein, Verhoeff's, and Weigert's elastic tissue stain. Of these a freshly prepared solution of Verhoeff's elastic tissue stain gave the best results.

The following four specimens of this type are presented, two of pig eyes and two of human eyes.

Specimen 1, Pig Eye (Figs. 1 and 2).—After preparation as noted above, slit lamp examination showed a smooth, mirror-like surface that had the contour of the posterior surface of the lens. Behind this was a narrow opaque zone, and behind this zone a space of varying width which was optically clear except for a small number of fiber-like structures; posterior to this the vitreous structure was seen. Microscopic examination shows a structure of definite thickness that stains faintly with hematoxylin and eosin, but takes a deep blue with Verhoeff's elastic tissue stain. The membrane extends from the position of the anterior portion of the pars plana ciliaris of one side to a corresponding position on the other side. All examined sections of an eye cut in serial sections showed this structure intact. A section of the lens shows the capsule intact.

Specimen 2, Pig Eye (Figs. 3, 4, 5, 6).—The eye was prepared as in specimen 1, and dehydrated through alcohols to absolute alcohol. The eye was cut anteroposteriorly, placed in absolute alcohol and ether, and then embedded in celloidin. Sections were cut in such a way as to show the membrane and cut edge of the vitreous in one section. This section shows a slightly wavy and wrinkled membrane extending forward and inward from a position slightly anterior to the ora serrata. The membrane ends sharply, and the faint edge of the cut vitreous extends posteriorly to the position of the optic disc. A section of the lens shows the capsule intact.

Specimen 3, Human Eye (Figs. 7 and 8).—Preparation was completed as in specimen 1. The slit lamp examination shows a smooth, even surface, with a curve corresponding to the posterior surface of the lens. Behind this is an opaque zone of irregular

thickness, on the posterior portion of which were a number of irregular deposits. The vitreous was fluid and contained floating granular opacities. The microscopic section shows a deep blue structure, extending from ciliary body to ciliary body, and slightly thicker at the periphery than in the center. The vitreous was granular. There was, however, no microscopic evidence of cyclitis. A section of the lens showed the lens capsule intact.

Specimen 4, Human Eye, Prepared and Examined as in Specimen 3.—In this specimen a folded and wavy membrane extended from ciliary body to ciliary body, and stained deep blue with Verhoeff's elastic tissue stain.

All of the eyes, with the exception of the two human eyes, in which a membrane was demonstrated, were normal and had been fixed shortly after enucleation. The first of the human eyes, specimen 3, was from a case of exophthalmos with well marked exposure keratitis. The eye was obtained twelve hours after death and immediately fixed. The vitreous in this case, as shown by slit lamp examination and by section of the fellow eye, was fluid. The other specimen was a normal human eye, obtained five days after death. This accounts for the marked granularity of the vitreous as seen in the histological preparation.

We believe that the evidence presented warrants the view that there is a non-nuclear, structureless, uniformly staining membrane that completely separates the aqueous from the vitreous chamber, is normally in contact with the posterior lens capsule, and which may be designated as the anterior hyaloid membrane of the vitreous.

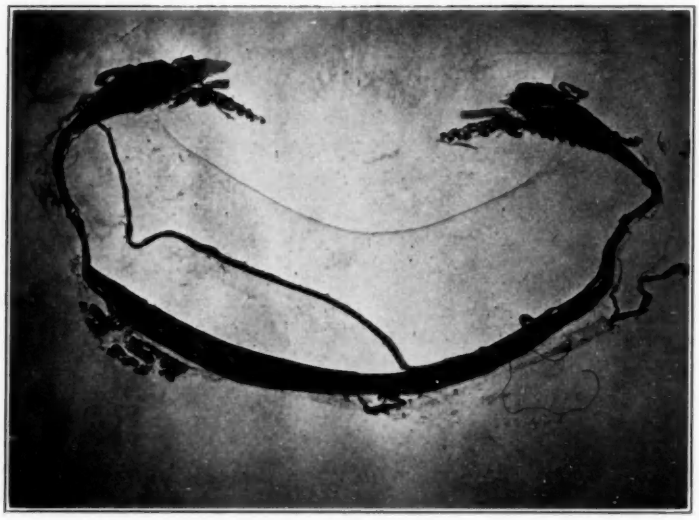


Fig. 1. Section through a pig eye with the cornea and iris removed, and the lens extracted in its capsule, showing the hyaloid membrane of the vitreous stained with Verhoeff's elastic tissue stain.

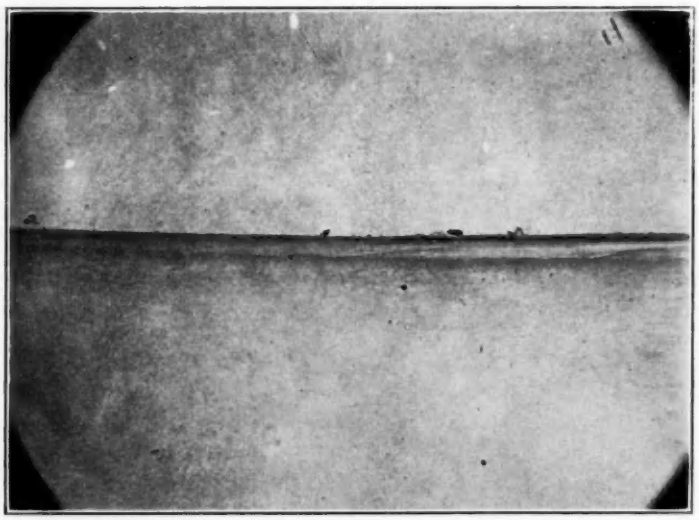


Fig. 2. The hyaloid membrane of the vitreous of the same eye (Figure 1) seen under higher magnification.

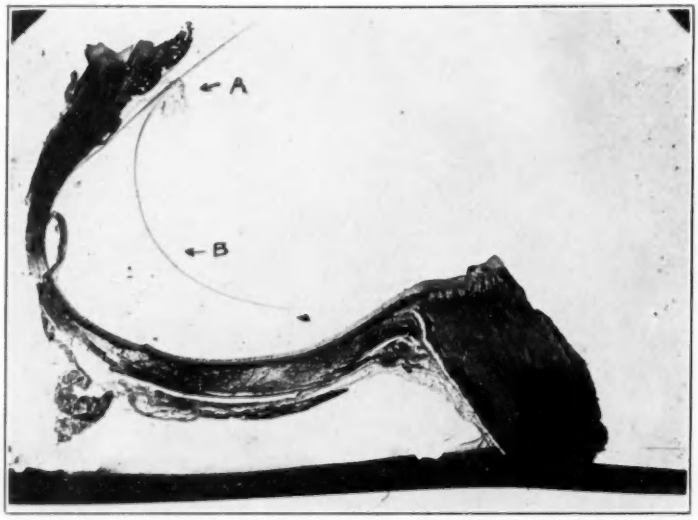


Fig. 3. Section of a pig eye cut to show the hyaloid membrane at A, and the sagittally cut edge of the vitreous at B.

Fig. 4. Higher magnification of the same eye (Figure 3), showing the hyaloid membrane at A, and the sagittally cut edge of the vitreous at B.

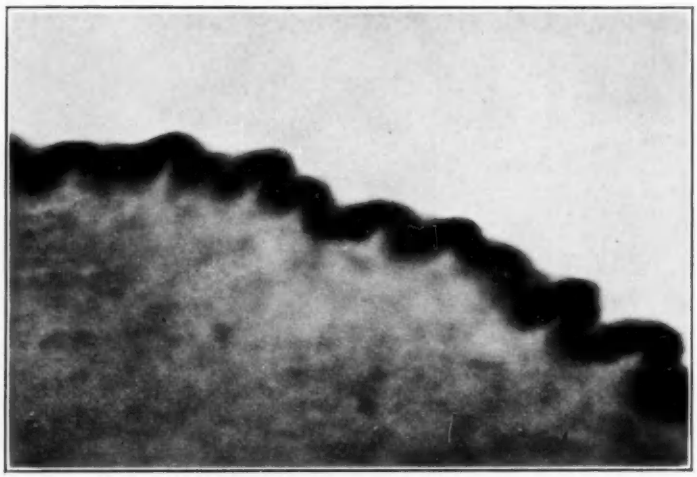


Fig. 5. The hyaloid membrane of the vitreous seen under high magnification.

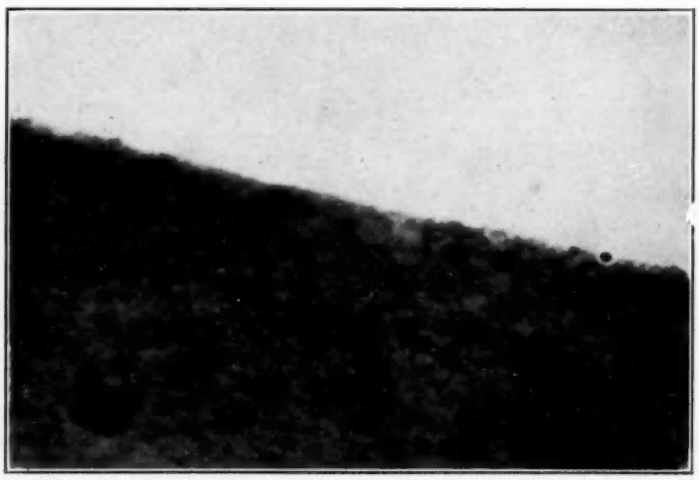


Fig. 6. The sagittally cut edge of the vitreous seen under the same magnification as in Figure 5.

Fig. 7. Drawing of the slit lamp appearance of the hyaloid membrane of the vitreous seen in a human eye after removal of the cornea and iris, and the lens in its capsule.

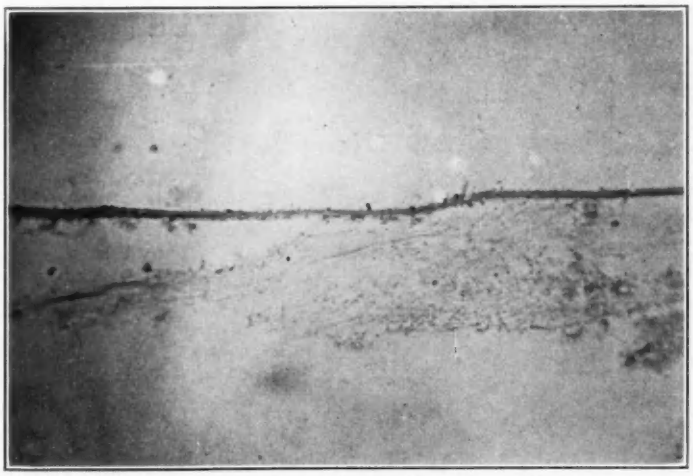


Fig. 8. Microscopic section of the hyaloid membrane of the vitreous of the same eye (Figure 7).

DISCUSSION.

DR. R. C. GAMBLE, Chicago: The question whether a true hyaloid membrane of the vitreous exists or is only an artefact produced by the action of fixing solutions on the fibrils in the vitreous, has for many years been an interesting subject for study and discussion. Dr. Cowan's observations with the slit lamp on human eyes after intracapsular extraction of cataracts is valuable evidence that such a membrane does exist. The later experiments described in this paper are, of course, open to the usual criticism, because it was necessary to use formalin fixation before the delicate dissections could be made.

Embryological studies indicate that the membrane covering the anterior part of the vitreous is a condensation of the fibrils between the primary and secondary vitreous, and gives no evidence whatever of a hyaloid membrane posteriorly. Histological examinations of human eyes prepared in the usual manner almost always show a membrane extending across the globe, back of the lens, as described by the authors, and careful observation will show that its anterior surface is smooth but its posterior surface is somewhat shaggy, due to small fibrils loosely attached to the membrane, and the whole membrane is definitely fibrillar. Also, the anterior part of the vitreous is especially rich in fibrils, and in this region they tend to be arranged parallel with the plane of the hyaloid membrane. This point is brought out especially in cases where the vitreous is filled with pus cells. In the anterior part these cells are often arranged in parallel rows between the fibrils, while in other parts of the vitreous there is no special arrangement of the pus cells.

My own feeling is that there is an anterior hyaloid membrane which is simply a condensation of the vitreous fibrils

DR. DANIEL S. KIRBY, New York City: I think Dr. Cowan and Dr. Fry are to be congratulated on their method of approach to this subject. I am very much interested in the matter of the hyaloid membrane in the human eye, and have made numerous observations. I will show some slides illustrating the appearance as seen with the slit-lamp microscope, and draw the following conclusions for you:

A study of the anterior vitreous of the human eye in health and disease was undertaken to correlate the existing knowledge concerning the so-called "hyaloid membrane" and "retrolental space," and to solve some of the disputed questions concerning structures and relations in this area of the human eye.

Embryological studies by Mann demonstrate that the anterior vitreous consists of the *primary*, which is directly posterior to the crystalline lens in the adult, and the *secondary*, which is separated from the primary by a condensation layer representing the wall of the canal of Cloquet.

Surgeons are familiar with a layer known as the "hyaloid," and anatomists have described it as being in apposition with the posterior lens capsule except for a capillary space. Doubt was thrown upon this conception by slit-lamp microscopists who found uniformly in normal eyes a retrolental space.

Examination of the vitreous with the aid of polarized light demonstrates that there is no structureless membrane in or surrounding the

vitreous. Ultramicroscopic examination led to the hypothesis that the vitreous is a gel without any real fibrous structure. The appearance of fibrillæ or curtains is the result of the superimposition of numerous ultramicroscopic micellæ. The "structures" or "curtains" that are seen in the normal vitreous are therefore condensation layers. The most anterior of these must represent the so called "hyaloid" and it is to be found directly back of the posterior lens capsule, separated from it only by a capillary space and kept in apposition with the posterior lens capsule by the intravitreous pressure.

Observation of the posterior lens capsule region, both directly and with the specular reflex, discloses a fibrillar layer immediately posterior to the capsule. The "arc line" and other remnants of the branches of the hyaloid artery may be found attached to this fibrillar condensation layer. They are only secondarily attached to the lens capsule through the intimate contact of the anterior condensation layer to the lens capsule at its periphery.

The visibility of the "outstanding beam" through the retrolental space proves that it is not optically inactive. The fact that the visibility of this beam is greater than that of the aqueous proves that it contains a colloid which is different than that of the aqueous humor. The latter as such does not exist in the retrolental space. There is no reason to believe, however, that the fluid which is in solution in the gel of the vitreous protein is of any other nature than that of the aqueous.

The retrolental dark space is proved to contain primary vitreous by demonstrating that it is the anterior portion of the canal of Cloquet. The "moire curtain" at the posterior wall of the retrolental space is none other than the condensation layer separating the primary and secondary vitreous.

Liquefaction of the vitreous undoubtedly represents a lysis of the colloidal gel. This may possibly be affected by an enzyme action producing digestion of the ultramicroscopic micellæ. The typical normal appearance is lost and there is substituted a condition in which one observes a variety of formations which represent most probably aggregations of the ultramicroscopic fibrillæ into dots, nodes and fibres of higher refractive index. They are observed as being suspended in a fluid of varying degree of viscosity. Floating membranes may thus occur without fibroblastic invasion of the vitreous.

Particulate bodies in the retrolental space behave as if enmeshed in a gel such as the vitreous is conceived to be. Such bodies, except for developmental remains, may always be considered abnormal and indicative of a pathological condition. In nearly every case of iritis, cells and debris may be found in the retrolental space simultaneously with their appearance in the anterior chamber. From this standpoint cyclitis undoubtedly is coincident with iritis. In cases of suspected cyclitis or uveitis the retrolental space should always be examined for cells. In choroiditis, cells may come from a posterior lesion through the vitreous to the retrolental space. Clearing of the retrolental space of cells and debris is undoubtedly accomplished in the same manner as that of the rest of the vitreous, by a process of lysis and digestion and phagocytosis by the tissue macrophages. In every case of detachment of the retina with tear, and in

those in which a tear was suspected but not seen, reddish brown lustreful pigment granules exfoliated from the retinal pigment epithelium of the area of the tear have been observed in the retrolental space.

After operations on the lens in which the posterior capsule-zonular structures are left intact, the relations in the retrolental space remain unaltered. After uncomplicated intracapsular extraction of the lens, there is to be observed a delicate condensation layer, so called "hyaloid," limiting the anterior vitreous, and an area that corresponds to the retrolental space. Posterior to this space the typical "moire" curtain that is normally seen at the posterior border of the retrolental space may be observed in a certain number of cases. In the elderly, partial liquefaction of the vitreous is common and normal appearances are for this reason not preserved in all cases after cataract operations. After discission of secondary cataract, division of the anterior condensation layer is the rule and herniation of the vitreous through the pupil is very common. Even in this condition there appears an impact line of separation from the aqueous produced by the difference in surface tension of the aqueous and vitreous.

The most important conclusions are that:

1. There is a condensation layer (hyaloid) representing aggregations of ultramicroscopic micellæ limiting the vitreous anteriorly.

2. This layer is in apposition with the posterior lens capsule except for a capillary space which contains a fluid presumably like the aqueous, but which is kept practically obliterated in the intravitreous pressure.

3. The retrolental space limited anteriorly by the anterior condensation layer and posteriorly by the wall of the canal of Cloquet contains primary vitreous.

Dr. Robert von der Heydt, Chicago: Dr. Cowan is to be congratulated on this report because his findings seem to reconcile two controversial conceptions. One of these is that the postlental space is but a capillary slit. The other, that it is quite deep and filled with a thin fluid vitreous, as may be proven by the microarc slit-lamp. Both of these ideas seem to be possible with Dr. Cowan's findings of a thin membrane adjacent to the lens.

I have studied membranes in eyes that have been operated for cataract by the intracapsular method. In these cases the membrane seen is not the posterior lens capsule. It can be a coagulum, as Dr. Gamble stated, but may, in my opinion, be the hyaloid membrane. I have seen similar membranes extend upward and join the extraction incision, thus coming anterior to the plane of the iris. It is possible that these membranes are drawn upward by the lens during the extraction and thus made adherent to the wound. Vitreous is apt to do this—it often follows the knife needle toward the wound and becomes attached to the latter. This is a common slit-lamp finding.

Dr. V. A. Chapman, Milwaukee: I have had a case just now that demonstrates very clearly to me, clinically, that there is a definite hyaloid membrane. I did a cataract operation in a comparatively young patient in which the lens itself was not opaque; it was definitely clear, but there was a streak across the posterior capsule which annoyed her so much that

she wanted it removed. I lifted the lens out without any trouble, and the slip-lamp showed a definite membrane entangled in the outer angle of the incision.

I believe one reason we do not see it more often is because usually the cataract is senile, of long duration, and there is attenuation of this membrane which has been considered degeneration of the vitreous itself. Personally, I am sure this membrane exists. Furthermore, it is not to be tampered with. It does become opaque on the surface.

Dr. Mark J. Schoenberg, New York City: Last winter I had the opportunity of demonstrating before the Eye Section of the New York Academy of Medicine a comparatively new way of studying the anatomy of the eye with the aid of the slit-lamp microscope. On that occasion I showed specimens of the vitreous. I have always endeavored to study the consistency and texture of the vitreous without committing any violence to that tissue, and of course I have avoided the use of alcohol and formaldehyd and other such substances.

I would like to ask Dr. Cowan whether he is absolutely convinced that there is a hyaloid membrane. It is true, I have also seen similar formations which looked very much like the specimens that Dr. Cowan presented here today. The main objection against these methods of study of the vitreous, my own as well as those of Dr. Cowan and his associates, is the violence we commit by placing the specimen in formaldehyd. If we avoid using hardening fluids we fail to find the membrane described by Dr. Cowan.

Furthermore, a strong argument in favor of the existence of a hyaloid membrane is his findings in patients after an intracapsular cataract extraction. Dr. Cowan has seen in such cases something that looks like a membrane prolapsing in the pupillary area. But I should like to ask whether, in cases in which there is an adhesion between the lens and vitreous after the lens is extracted in capsule and the vitreous has thereby suffered a tear, does one find a pellicle-like hyaloid membrane hanging loosely in the aqueous? No. All one sees is strands of vitreous floating in the vitreous. No membrane.

I do not say that I deny the existence of hyaloid membrane, nor do I say there is one. The incontrovertible evidence of a hyaloid membrane, its anatomical demonstration, its specific staining, and its separate pathology, are still awaiting a demonstration.

Dr. Alfred Cowan, closing: I am glad that Dr. Kirby agrees with what I have always held; that is, that the retrolental space has no direct communication with the posterior chamber, although this is contrary to the opinion of many investigators. As a matter of fact, I think there are definite reasons for my belief. On the other hand, if this membrane, which we consider the hyaloid, surrounds the vitreous entirely, then the vitreous must, of course, be separated from the posterior chamber.

Dr. Chapman mentioned opacities of the posterior surface of the lens. In a small book by Captain Surgeon Fink, published in 1894, on The Extraction of the Crystalline Lens in Cataract, he mentioned the very thing of which Dr. Chapman speaks. He, Fink, considered that many opacities that were supposed to be in the posterior portion of the lens or

at the posterior capsule, were really in the hyaloid membrane and not in the lens at all. Immediately after operation he examined all his cases of cataract extraction with an ophthalmoscope and found many of these opacities entirely outside of the lens. There is no doubt that we do have opacities of the hyaloid.

Millette reported a series of after-intracapsular lens extractions, and he found in several cases a membrane that was opaque and which did not respond to treatment. When discission was done in this secondary cataract, the opening immediately closed. I think that this was nothing more or less than an overgrowth or abnormality of the hyaloid membrane, and the reason for the opacity in the first place was the reason why it did not respond to treatment.

The objection which was made in regard to the hardening process in the preparation of our specimens does not seem pertinent. We made every effort to show that these processes had no effect on the cut vitreous. The enlarged photograph seems to prove this to us.

In response to the remarks of Dr. Schoenberg, I should like to say that in several eyes, after removal of the cornea, the iris, and the lens, an incision was made with a small knife, while the result was observed with a slit-lamp and corneal microscope. Through the incision we could see the dark, transparent vitreous come forward and the edges of the wound, which were gray by comparison, fold outward or inward. It seems that nothing but a membrane could act in this way.

OTOLARYNGOLOGIC SESSIONS

THE PROGNOSTIC SIGNIFICANCE OF SINUSITIS IN CHILDREN.

LYMAN RICHARDS, M.D.,

BOSTON.

The practitioner of medicine, like many a worker in less altruistic fields, cannot pursue the even tenor of his way without encountering, from time to time, waves of enthusiasm which center about and emanate from some increased interest in a particular disease entity. This interest arises in the minds of a few choice souls who, as pioneers in all medical progress, first broach the subject matter to their confreres at some meeting, with the hope that the latter may be persuaded to go back into their communities and preach the same gospel. This they proceed to do, and if the idea under discussion be at all readily grasped by the laity it is but a short time before the disease becomes in many respects a popular one, discussed, even to some extent in technical terms by the public, featured by the press, and finally viewed as almost a requisite for social equality. Such a disease some years ago was appendicitis, and the removal of the offending organ was considered as a sort of badge of entrance into the band of the surgically elect.

More recently, in the field of laryngology, the removal of the tonsil has had a vogue beyond all reason, but a vogue which still continues to the present day. The public have become so educated to the idea of tonsil disease in itself and as a cause of unrelated symptoms and to the operation as a cure-all for an almost unlimited variety of complaints that they make their own diagnosis, ofttimes before reaching the laryngologist's office. Not one of us but in clinic or consulting room, on inquiring as to a patient's chief complaint have been greeted with the reply "Tonsils, doctor," with an air of finality that precluded in the patient's mind any further need for inquiring into the symptomatology, leaving only the formal conclusion of the arrangement for the operation.

That such an attitude, with its resultant surgical abuse and unavoidably disappointing results must be combated by sane and level headed practitioners of medicine goes without saying, and already there are signs that, so far as the tonsil question is concerned, the pendulum is beginning to swing less violently—in fact, a little even in the other direction. Particularly the pediatricians are protesting against indiscriminate operation on children until complete physical examination and laboratory studies have eliminated all but the tonsil as the probable source of trouble. One even finds a pediatrician from time to time who rather prides himself on advising tonsillectomy only for the most urgent and indubitable indications.

In the gradual checking of such a public stampede, and once started, no matter for how good a reason, it is hard to check, there rise up from time to time some individuals who for one reason or another are peculiarly able to contribute data which, by virtue of its comprehensiveness and the period of time covered by it, is of the utmost value in throwing much needed light on such problems. Such data must be based on as large a number of patients as possible, and if the advisability of operation is in question, should include patients who have not been operated upon as well as those who have, comparing their symptoms before and afterwards. When such statistics are carefully compiled and compared, we come nearer to being able to estimate the true value of a procedure or the status of a disease entity than in any other way. It was in this manner that the incomparable statistics of Kaiser of Rochester did so much to illuminate the tonsil question and provide more dependable criteria than have heretofore been available.

And now within the last ten years there has appeared for recognition by the public a new disease, sinusitis. The office patient and, to an ever increasing extent, the clinic patient, replies to the interrogation as to the difficulty at hand, "Sinus, doctor." So accurate has the lay diagnosis become that the patient can differentiate between a pain over the eye which is a "sinus" and a pain over the antrum which is not. From a medical point of view, sinus disease has been brought into prominence as a cause of almost as many different symptom complexes as were once attributed to the tonsil, and in many instances rightly so.

Particularly has this been the case with children. Up to the time when Dean first published his work on sinusitis in children

the entire subject had received but meager attention, and one shudders to think of the variety of conditions which were forced to masquerade under some other etiologic title. Gradually the propaganda in regard to sinus disease in children has spread until today it is universally recognized and considered as a diagnostic possibility or excluded as a cause of a considerable number of pediatric conditions. And so the question naturally arises, are we again, as in the case of the tonsil, on the verge of letting our enthusiasms run away with us, of thinking too easily and exclusively of the sinus as the etiologic factor in a variety of symptom complexes, and are we in need of at least pausing for a moment to consider the situation as it exists with respect to children today in the hope of being able at least to assure ourselves that we are on the right course?

Having felt that such a taking account of stock of the sinus question would be of interest and, it is hoped, of some value, I have undertaken to examine the records of some five hundred cases of sinusitis which have been treated at the Children's Hospital in Boston during the past fifteen years. The records include the days when the diagnosis of sinusitis was almost unknown and those in which it appears constantly. They include every form of the disease, from the most acute and fulminating, to the mildest and most chronic. They include every form of treatment, from the most radical surgery to the most inadequate and neglectful medical procedures, for which latter in his earliest days the present head of the laryngologic department takes all the blame. They include, with varying degrees of completeness, an account of the child's subsequent course and his present state of health. For these facts I have been forced to rely, first on the hospital records themselves, which were not always as complete as could be desired; second, on questionnaires sent to the mothers of patients, and third, on X-ray films taken of children who were brought back to the hospital for this purpose.

This data can in no way compare in completeness and accuracy with the above mentioned statistics of Kaiser on the tonsil question. Nevertheless, covering, as they do, inadequately, the ten-year period during which the subject of sinusitis in children has come into such prominence, they will, I believe, furnish some information which will serve to stabilize the question and enable us to proceed in the treatment of sinusitis with more surety and success.

Information has been sought in the above manner on the following points:

1. The course of the nasal disease since the diagnosis of sinusitis was first made. Has it subsided, either with or without medical treatment; has there been recourse to surgery, and what has been the symptomatic end result from the nasal point of view?

2. The status of the sinus itself, as revealed by an X-ray check-up. What is the relationship between the symptoms and the condition of the sinus as evidenced by this X-ray?

3. Some estimate as to the value of different types of treatment, conservative or radical, in respect to the end results.

The data comprising this survey consists of 480 hospital records, 150 questionnaires returned by mothers of patients and 75 X-ray check-ups of the sinus condition at the present time. It is of course to be regretted that there is not available a questionnaire and an X-ray for every patient. Questionnaires were sent to each patient, but many of them could not be located by mail and many failed to return any reply. Although almost every parent expressed a willingness and desire to have, without expense to them, an X-ray check-up of the child, only a small percentage took the trouble to call up for an appointment. Conclusions, therefore, must be drawn from data of different values, depending on the completeness of the records. Often only portions of the questionnaire were answered, and sometimes an adjoined letter attempted to cover the matter independently. There were surprisingly few returns which contained any reproachful criticisms where treatment had obviously been unsuccessful, and expressions of appreciation of the interest shown by the questionnaire were numerous.

The extraordinary increase in either the frequency of sinusitis or its consideration as a diagnosis is at once evident on examining these records. In 1918, out of 2,700 patients treated in the otolaryngologic department the diagnosis of sinusitis appears only four times, while in 1930, out of 7,200 cases, it appears 266 times, a percentage ratio of 1 to 23. It is hard to believe that the disease itself is twenty-three times as common as it was twelve years ago, and one must conclude that the increase in discussion of the subject and the greater alertness in making the diagnosis is responsible for these figures. The intervening years show a proportional change in the same direction.

The series included only records in which an X-ray showed definite evidence of involvement of the maxillary antrum in one or both sides. While by no means the infallible sign of sinusitis, it serves as the most satisfactory and constant criterion by which to estimate the existence of this condition. There were, of course, on this basis all varieties and degrees of involvement, ranging from a mild and transient obscuration to an almost complete opacity, and the results of any form of treatment would vary in respect to the degree of pathology present. What I have sought here, however, has been a glimpse of these cases as they come and go, and some generalized information as to the outcome of them under different forms of treatment.

The series includes very few cases under two years of age, sinusitis occurring predominantly among the children between five and ten. Sixty per cent were boys; 34 per cent were girls.

Treatment was carried out either in the hospital wards or in the outpatient department. The former proved invariably far more satisfactory for investigation of these patients and for the securing of satisfactory therapeutic results, but unfortunately it is impossible to hospitalize all these cases and they must be attended in clinics as well as possible. Only in so far as more and more thorough study is made of these patients in hospital wards will progress be made in solving the still perplexing problems surrounding this condition of sinusitis.

The cases have been divided into two main groups, the one comprising the acute, the other the chronic form of sinusitis. By acute is meant the early more or less purulent form of infection, such as is associated with pyogenic organisms, with or without actual pus formation. The chronic type is characterized by a thickening of the lining membrane, with little or no purulent secretion, is often allergic in origin, and often only one factor in a bizarre symptom complex. The acute and chronic forms of sinusitis not infrequently overlap, but nevertheless are in most respects sufficiently distinctive to warrant this subdivision. In this series 30 per cent were included as acute and 70 per cent as chronic sinusitis.

I propose to consider the subject of sinusitis in children principally from the prognostic standpoint. Given a known condition of a sinus and a known form of treatment, what may be anticipated as to the outcome, symptomatically and functionally? Can the initial condition and the treatment of it be correlated with

the end result and therefore some conclusions be drawn as to the efficacy of a given treatment in a given type of sinusitis?

Naturally such conclusions will be more dependable in the records in which a questionnaire has been returned as to the present state of the child's health, and still more so in those in which X-ray check-ups add further data. Some conclusions can, however, be drawn from the records which lack this confirmatory information, and these I propose to examine first, supporting them by facts furnished by the other supplementary data.

A. Data obtained from examination of hospital records during the past twelve years, where parents returned no questionnaire and did not return the children for X-ray examination:

In classifying these records I have attempted to estimate the results of the treatment which these children received by dividing them into three groups: those who appeared to have been cured, those who could fairly be said to have improved, and those who did not show any improvement. These estimates were often difficult to arrive at, since the records in many instances did not specifically state the final end result, and much reliance had to be placed on the progress notes and reports from other departments in the hospital. Nevertheless, I believe that some information can be derived in this manner which will shed light on the prognosis of sinusitis as it is seen in a large children's hospital.

I shall consider these cases by dividing them into acute sinusitis and chronic sinusitis, and further subdividing them according to whether they received principally medical or surgical treatment. By medical treatment I understand the use of such procedures as nasal irrigations, instillations of various medicinal drops, sprays, suction, inhalations and finally medical procedures carried out in other clinics, such as the anaphylactic and allergic. It is impossible to list these cases separately according to the individual type of medical therapy, but they are classed as a group in contrast to the surgical cases. The treatment which these latter received has been grouped under three main headings: (1) Removal of tonsils and adenoids; (2) antrotomy (intranasal, with or without irrigations); (3) middle turbinectomy, and (4) radical surgery. It must be understood that there were frequent instances where there occurred various combinations of these procedures and also in which medical and surgical treat-

ment were combined, but so far as possible information has been sought on the results of a single form of therapy.

Fig. 1 indicates the situation as it pertains to the cases classified as chronic sinusitis. Medical and surgical therapy are contrasted in respect to their end results and the percentages in each group are recorded. Several interesting facts are brought out in an examination of this chart.

(a) Medical treatment alone resulted in a disappointingly small percentage of cured cases and a surprisingly high number of failures. One can only conclude that some form of surgery or systemic investigation must often be resorted to in order to reach the fundamental difficulty whereby alone a complete cure

Fig. 1. CHRONIC SINUSITIS.

(General Records without Questionnaire.)

Result	Medical Treatment	Surgical Treatment			
		Tonsillectomy	Turbinectomy	Antrotomy & Irrigation	Radical Surgery
Cured	20%	33%	40%	14%	16%
Improved	42%	66%	50%	75%	65%
Unimproved	38%	1%	10%	11%	19%

is attainable. Medical treatment, however, achieved improvement in a considerable number of instances.

(b) Surgical statistics show a fairly satisfactory situation in regard to the removal of tonsils and adenoids, and the partial removal of the middle turbinate. The former has long been advocated as a most important measure in the treatment of sinusitis, and with its high percentage of improvements and total absence of failures thoroughly justifies itself. Turbinectomy has been less in vogue and might even evoke some adverse criticism, but statistically it has been quite successful, presumably because it relieves a chronic congestion around the natural ostium and thereby permits restoration of normal aeration of the sinus. Antrotomy and radical procedures made a good showing only as

they served to bring about an improvement, but resulted in only a few cures and only a small number of complete failures.

Fig. 2 indicates the situation in regard to acute sinusitis. Here again medical therapy appears to have been able only to effect a considerable degree of improvement, cures and failures being about equal. From the surgical aspect tonsillectomy appears to have been even more succesful than in the chronic cases, as does also antrotomy, a procedure apparently more adapted to acute than chronic cases. Turbinectomy does not figure in the acute statistics. It will be noted that radical surgery proved extraordinarily unsuccessful, not because of any intrinsic deficiency but because it was reserved in almost all instances for the very acute fulminating types of infection in which the risk and probable

Fig. 2. ACUTE SINUSITIS.

(General Records without Questionnaire.)

Result	Medical Treatment	Surgical Treatment		
		Tonsillectomy	Antrotomy	Radical Surgery
Cured	23%	72%	57%	30%
Improved	50%	18%	43%	10%
Unimproved	27%	10%	0%	60%

mortality would be likely to be high in any case. It was in this group that there occurred the six deaths which were found among 480 cases. They all showed the typical picture of swelling of the eye and surrounding orbital tissues and purulent infection of adjacent sinuses such as the ethmoid and frontal. With one exception these fatal cases were all operated upon in a more or less radical manner, either externally or under the lip, and they all developed some complication such as osteomyelitis, septicemia, meningitis or brain abscess. They were all marked at the onset as of unusual gravity by the external swelling of the soft parts, distinguishing them from the ordinary type of purulent sinusitis. They form a fortunately small group which must be encountered from time to time and which calls for keen judgment and courage

in its treatment. My conclusions have been that the longer radical drainage can be withheld with any reasonable safety, the better will be the outcome. Some form of radical surgery will almost certainly have to be undertaken sooner or later, but at the outset every encouragement to nature to wall off the process must be afforded, even in the face of high fever, delirium and intense toxemia. Serious consideration should be given two-stage operations with simple drainage of purulent foci with later radical extirpation of disease. In any case, the mortality will be higher than in other forms of sinusitis.

In reviewing these records, one is impressed with the extraordinary complexity of the situation. In addition to their sinusitis these children often were under treatment for other diseases which may or may not have had a bearing on the sinus condition. This treatment was frequently shifted from one form to another, Their living conditions and diet and habits of work and play were a constant factor, and the element of allergy was often creeping in to complicate matters. One is left with the impression that unless a child can be isolated and controlled in respect to its activities, its environment and its treatment, sinusitis will remain a difficult problem to solve.

B. Data bases on 150 questionnaires returned by parents. The information asked for was as follows:

1. Have you taken the child for treatment of his nose to any other hospital since your last visit to the Children's Hospital?

2. Has the child had any further trouble with the nose or sinuses since your last visit to the Children's Hospital? Is the child subject to head colds or running nose?

3. Has he had any other illness since then?

4. Has he any trouble at present with his nose?

5. Would you be willing, without expense to yourself, to have the sinuses X-rayed in order that I may check up their condition at present?

I sought information on the question of visits to other hospitals in order to check up the cases which had not been back to us for a considerable period of time. It was somewhat of a pleasant surprise to find that only 12 per cent reported having sought treatment elsewhere, and this did not always indicate dissatisfaction with the treatment received, numerous other reasons, such

as "over age" or "leaving the city," being assigned as the explanation. Any condition requiring repeated visits to a clinic must necessarily result in some loss in clientele, no matter what pains are taken to avoid it.

Based on the replies to questions 2 and 4, data has been classified to indicate the results of treatment received by the group of children concerning whom the parents supplied up to date and presumably fairly accurate information. Doubtless inaccuracies have crept in, and the decision as to whether a given case has been cured, improved or unimproved by treatment had at times to be somewhat arbitrary. Nevertheless, this method of arrangement has been pursued as being, on the whole, the most practical and satisfactory. Certainly these figures are far more accurate and conclusive than those obtained from an estimate of the hospital records alone.

Fig. 3 gives the status of medical treatment as contrasted with that of surgical treatment, the latter being a composite figure made up from the various surgical procedures carried out. Here it will be seen that, contrary to the figures obtained from the hospital records, medical therapy has a better record than its surgical rival with more cures and fewer failures. This discrepancy is somewhat hard to explain, but may be due to unavoidable errors associated with drawing conclusions from the records alone.

Fig. 3. RESULTS OF TREATMENT.
(By Questionnaire.)

Type of Treatment	Cured	Improved	Unimproved
Medical	44%	17%	39%
Surgical	34%	20%	46%

Fig. 4 is an analysis of the end results according to whether patients were treated medically or surgically. Of the cured cases a preponderant part were given medical therapy. In the improved and unimproved groups the results were about equally divided between the two forms of treatment. The total number of cases treated, either medically or surgically, was almost equal.

Fig. 4.

Result	Surgical Treatment (46% of Total)	Medical Treatment (54% of Total)
Cured	37%	63%
Improved	48%	52%
Unimproved	47%	53%

Fig. 5 is a tabulation of end results according to whether the infection was an acute or chronic one. Medical therapy was apparently much more successful in dealing with the chronic infections than was surgery, and surgery much more potent in curing the acute conditions. This is in accord with general principles in which surgical drainage is consistently effective in dealing with purulent infections. Regarding the cases which failed to improve, it is apparent that these fall to a very great

Fig. 5. END RESULTS BY QUESTIONNAIRE.
(SYMPTOMATIC.)

CURED		
Treatment	Acute	Chronic
Medical	10%	90%
Surgical	45%	55%

IMPROVED		
Treatment	Acute	Chronic
Medical	44%	56%
Surgical	44%	56%

Fig. 5. END RESULTS BY QUESTIONNAIRE.—Continued.

	UNIMPROVED	
Treatment	Acute	Chronic
Medical	18%	82%
Surgical	30%	70%

extent among the chronic group and offer a far more stubborn problem than do the acute cases. So far as improvement went, there was not much to choose between either type of treatment.

Fig. 6 is designed to show, regardless of end results, the division of treatment of the acute and chronic cases. As might be expected, a larger percentage of the acute cases were treated surgically and a larger percentage of the chronic cases treated medically.

Fig. 6. TREATMENT.

Type of Case	Medical	Surgical
Acute	39%	61%
Chronic	60%	40%

Fig. 7 is a composite chart of end results, regardless of what form of treatment had been received. The striking fact here is the high percentage of cases which had to be classified as unimproved, a failure in which both medicine and surgery shared about equally. It is in this fact that the greatest problem lies in dealing with sinusitis. In a large number of children local treatment of the sinus will not suffice to eradicate the symptoms, at least for any permanent period. Hence the conclusion is forced upon us that in many instances we are dealing with some systemic disturbance of which the sinusitis is only one local manifestation. Whether this systemic disturbance is in the nature of a true allergy or whether it is an endocrine dysfunction is beyond the

Fig. 7. END RESULTS BY QUESTIONNAIRE.

(SYMPTOMATIC.)

Result	OXO
Cured	39%
Improved	18%
Unimproved	43%

scope of this paper to discuss, but it must assuredly be recognized that sinusitis, and especially chronic sinusitis, is many times far more than a disease of the sinus.

A word in this connection as to asthma. Asthma was a complicating disease, almost always present at the time of the original X-ray investigation, in a surprisingly large number of patients with chronic sinusitis. These patients were most of them being treated in anaphylactic clinics and were referred to the laryngologic clinic for sinus investigation. Efforts to untangle the relationship between the sinusitis and the asthma will, I believe, continue to occupy many of us for some time to come.

Certainly what may fairly be termed routine outpatient treatment will continue to prove unsatisfactory and unproductive of ideal results. Until searching studies are made into the biochemical and physiologic activities of these patients we will fail to understand the underlying factors in this all too prevalent condition.

C. Data obtained through X-ray examinations of the sinuses at varying intervals after the known existence of pathology in these sinuses:

We come now to what may be called the more truly scientific portion of the material studied in this group of cases. Though comprising a regrettably small portion of the total cases, these X-ray check-ups of the condition of the sinus at the present time furnish a more accurate estimate of the local situation than can any statement of the parent or patient. It further affords an

opportunity to correlate the symptoms as reported by the questionnaire with the findings as shown by X-ray. In each instance these findings were compared with those noted on the first examination and the progress classified under three headings: (1) No change in the pathology; (2) improvement, and (3) complete return to normal radiability.

Fig. 8 represents the results of these X-ray examinations in respect to this classification. At once there appears the striking fact that in one-half of these patients there has been no improvement in the condition of the sinus so far as it may be judged by the X-ray. Almost all of the remainder, however, have cleared entirely, a small number only showing improvement.

It so happened that almost all of the cases returning for X-ray examination had been originally classified as chronic. This may possibly be due to the fact that the chronic cases proved so much more resistant to treatment and therefore the parents were more ready to co-operate in any suggestion that might lead to recovery of the patient.

Fig. 8. X-RAY FINDINGS.

(CHECKUP.)

Condition of Sinus	
Clear	41%
Improved	10%
No Improvement	49%

Fig. 9 shows the division of the cases checked by X-ray into those treated medically and those treated surgically. Since a greater proportion of the cases were treated medically than surgically in the entire group (54 per cent medical, 46 per cent surgical), it is apparent from this chart that there was no outstanding advantage in either form of treatment so far as its effect on the X-ray picture went. The two types of therapy maintained about the same relation, whether the X-ray showed complete clearing, an improvement or no change.

Fig. 9. X-RAY FINDINGS.
(CHECKUP.)

Treatment	Complete Clearing	Improvement	No Improvement
Medical (Chronic Cases)	61%	57%	61%
Surgical (Chronic Cases)	39%	43%	39%

Fig. 10 is a striking comparison between the situation as stated on the questionnaire and the findings reported by X-ray. It will be noted that cases reported symptomatically cured were to a large extent found to be cured by X-ray. Those improved in the one respect were likewise improved in the other, and the bulk of the patients who were unimproved symptomatically were found to have a sinus which showed no improvement by X-ray. One would, therefore, be justified in concluding that so long as there is X-ray evidence of pathology, the clinical symptoms of nasal congestion and discharge will similarly persist. Most of us have, I think, had the feeling that a sinus once subjected to chronic inflammation might, even in the absence of symptoms, fail to show a return to its original clarity by X-ray without indicating the need of any special treatment. While this is unquestionably true in many instances, it would appear from

Fig. 10. X-RAY FINDINGS.
(CHECKUP.)

	Cured (symptomatically)	Improved (symptomatically)	Unimproved (symptomatically)
Clear by X-ray	43%	40%	17%
Improved by X-ray	0%	62%	38%
No improvement by X-ray	21%	17%	62%

these figures that there is a tendency for the two factors to maintain a close correspondence with each other.

It will be noted on the questionnaire that information was requested as to the frequency of head colds after any form of sinus infection. While a certain number of head colds are almost a routine situation with any child, it is generally considered that the existence of sinusitis predisposes the individual to a greater than ordinary number of such infections. Sixty-four per cent of the patients reported being, as they usually put it, "subject" to head colds, while 36 per cent said they were not. A preponderance of almost two to one in this respect would lend some justification to the conclusion that sinusitis certainly carries with it the probability of an increased susceptibility to nasal infections. Such data should, of course, be checked by a comparison with the number of colds occurring in a group of children who had presumably never had sinusitis.

A word as to tonsillectomy. In the entire group of cases there were 62 per cent who had had their tonsils and adenoids removed before the recognition of the sinusitis. The therapeutic effect of the operation after sinusitis had developed has already been referred to. Certainly the operation will not ward off the occurrence of sinus infections, though these may well be less violent and of shorter duration because of the previous removal of infected lymphoid tissue.

What, then, are we to conclude to be the prognostic significance of sinusitis in children? If the inflammation is pyogenic in origin, is treated promptly, either medically or by conservative surgical measures, the condition will in general subside without leaving after effects that will be detrimental to the general health beyond a greater tendency to recurrent nasal infections. If, however, the infection progresses untreated, to a chronic state, or if, particularly, the sinusitis is but a local manifestation of general systemic allergy or other dysfunction, then the sinus disease will persist and continue to occasion local symptoms until the underlying cause is discovered and eradicated.

SUMMARY.

1. Sinusitis in children has become during the past ten years a well recognized entity, underlying local nasal symptoms and contributing to systemic disturbances in a relationship formerly never dreamed of.

2. Certain types of sinusitis are amenable to local and, particularly in the acute cases, surgical treatment.

3. Many cases of chronic sinusitis will defy all local measures, whether surgical or medical, and can only be relieved by investigation of the underlying physiologic and biochemical dysfunctions which exist in the individual patient. Allergic disorders will be found to play an important rôle in this connection.

4. In general, the existence of nasal symptoms will be found to parallel the existence of a pathologic state in the sinus as evidenced by X-ray.

5. Only by study and investigation with every clinical and laboratory facility at hand, principally in a hospital, can adequate study of these conditions be satisfactorily carried out.

319 Longwood Ave.

DISCUSSION.

Dr. Arthur W. Proetz, St. Louis, Mo.: It is a little difficult to discuss orally a statistical report of this kind. My own reaction to Dr. Richards' figures is that I hope he will carry the work of interpretation a bit farther; not from the standpoint of numbers—it must have been a laborious task to examine these 480 cases carefully—but from the standpoint of their true significance.

When we analyze figures of results under surgical treatment, for instance, we must remember that they are modified by the individual judgment of the surgeon—in other words, one operator with keen judgment of surgical indications would undoubtedly have a higher percentage of cures than another whose selection was less careful. For instance, regarding the turbinectomies, 40 per cent of those turbinectomized were cured, 50 per cent of those turbinectomized were improved and 10 per cent were not improved. These figures should be balanced against the observations of the same operator upon unoperated cases in which the indications were the same as for the operated ones. I feel that the analyses should be carried a little further before they are set down in black and white.

I am not overcritical but a little fearful that statistics such as these, not intended by the author to be final, will nevertheless be widely quoted to substantiate almost any contention.

I should like to learn whether the patients who had tonsillectomies and those who had turbinectomies were so treated because of diversity of opinion between two men attempting to cure the same condition or whether one man would have resorted to one treatment in one case and another in another because of diversity of symptoms.

I should like to hear of those children who, after a certain amount of medical treatment, had no relief and who were then submitted to surgical interference and observed for another year. It is often misleading to analyze hospital records of patients not personally seen because one may

find on closer analysis that the personal element in diagnosis, treatment and final evaluation has a considerable effect on the figures.

Another thing is the discrepancy between the figures deduced from the questionnaires and those in cases from which questionnaires were not returned. In regard to the relative efficiency of medical and surgical treatment, I wonder whether the effectiveness of the medical treatment in that group which replied to the questionnaire may not be explained by the fact that these people were more careful of their children. Those parents who complied in returning questionnaires probably co-operated best in pursuing home treatment. It is interesting also to speculate upon the 50-50 proportion between cured and unimproved patients. Let us compare these with a group of untreated patients to determine whether the results merely follow the law of averages or whether they have been influenced by some, or any, treatment.

One word about the X-ray: we see here that 62 per cent of those improved after operation showed improvement roentgenographically. In the unimproved group, the X-ray was correct again in 62 per cent; in the group of cured cases also a two-to-one proportion existed. Let these tables remind us that in one case in three, therefore, the X-ray is in error.

I wish to express my personal appreciation to Dr. Richards for undertaking this survey. It is a piece of work almost as thankless as it is important, and I hope he will go on with it to the extent of collecting data from others, as Mayer did with accidents from local anesthesia and Loeb with deaths from minor operations. The ultimate value of statistics as wide in their scope as these lies in the correlation of observations from many sources, and I hope that Dr. Richards will do this.

Dr. John J. Shea, Memphis, Tenn.: The sinuses reflect in their growth the infections they have experienced.

A severe infection before the migration of the fronto-ethmoidal cell (two years) may arrest sinus development.

With these facts as a working basis we use the size and stage of development of the sinuses as a criterion for our prognosis.

A composite picture of 100 normal sinuses, taken posterio-anteriorly in the 23 degree angle, shows the sinuses under two years to have the following characteristics:

(a) There is no frontal sinus.

(b) The ethmo-frontal bud is barely perceptible.

(c) The floor of the antrum is above the level of the floor of the nose.

By puberty (a) the frontal sinus has migrated and pneumatized to such a size that its breadth and height are equal, and the presence of partial septa have removed its cul-de-sac outline.

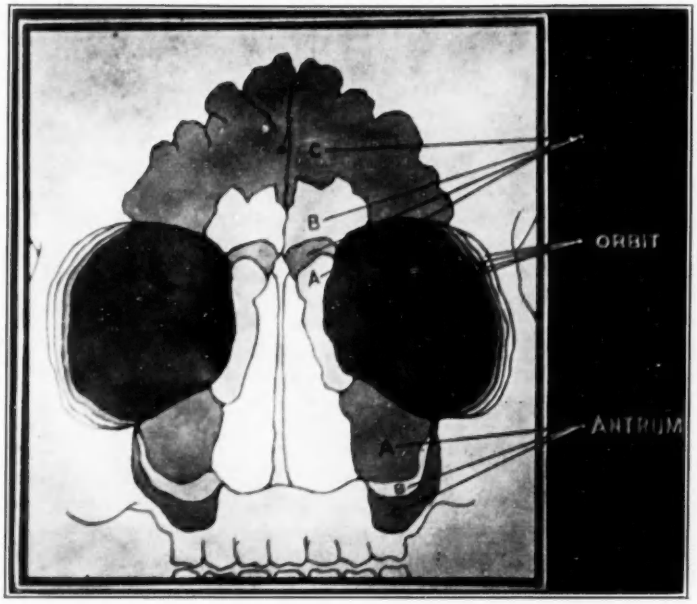
(b) The floor of the antrum is on a level with the floor of the nose.

The adult sinuses should show (a) matured frontal sinuses whose size depend upon family and racial facial features.

(b) The floor of the antrum is lower than the nasal floor and all the upper molar teeth have reached their position within the mouth.

A sirgle severe infection can cripple the sinuses as badly as many mild attacks.

The re-establishment of growth is a good prognostic sign.



A composite picture of 100 normal sinuses taken posterio-anteriorly in the 23 degree angle.
A—Before 2 years of age.
B—Puberty.
C—Complete.

A roentgenogram was shown of a girl of five who had a persistent infantile set of sinuses, the result of acute infections before two years of age. This child spent a spring in the sunshine of Florida and with prolonged local treatment, including antral irrigations, now presents a roentgenogram of sinuses equal or better than a child of her eleven years.

The re-establishment of growth with its spurt to normal puberty size required the curing of the sinus disease. This is attested by the fact that this girl lost only one day out of the last two years of school.

Dr. A. M. Alden, St. Louis: It is a rather refreshing thing to find 500 cases of sinus disease in Boston. I did not think they ever had them there.

The classification that Dr. Richards has made of acute and chronic sinusitis in children seems to me to be very apt, because my own feeling is that these are almost two separate and distinct clinical entities as far as the child is concerned. In other words, that the chronic disease is not simply a prolongation of an acute sinus infection. An acute sinus infection is an acute suppurative process involving one or more sinuses and, as a rule, it is not the sinusitis *per se* that needs surgical treatment, but

the complications of this acute condition, and such surgical treatment must be as radical as the given case needs.

Chronic sinusitis is the problem that we are all concerned with today. Personally, I have no conclusions about this condition at all. My mind is really in a very chaotic condition. I have been treating these cases for years, considering them perhaps largely surgical, and getting results, some of which I was not particularly proud. The more of these cases I see, the more I am convinced that a large percentage of chronic sinusitis in children is merely a local manifesation of some chronic dyscrasia in that child. The majority of these cases in my hands in the last two years have done much better under medical than surgical treatment. I am trying to get a better understanding of the fundamental physiology of the nasal mucous membrane in its relation to the other tissues of the body, a better understanding of the interchange of fluids between mucous membrane and other tissues, and perhaps a more adequate comprehension of some of the fundamentals of proper nutrition for the child.

I should like to ask one question of Dr. Richards. What do you consider the curative value of the adenoidectomy in these cases? In the majority of hospitals, tonsillectomy in children is usually associated with adenoidectomy—spoken of as T. and A. My own feeling is that in a great majority of cases an adenoidectomy properly performed is of much more value than a tonsillectomy, and I wonder, in these cases of Dr. Richards, what part tonsillectomy or adenoidectomy played.

Dr. M. F. Arbuckle, St. Louis, Mo.: Dr. Richards is trying to solve a problem, or a set of problems, which have afforded me and, I daresay, many of us, difficulties which, to put it mildly, are yet to be overcome. I admire his ingenious method of attack and his unusually frank statement of results obtained by treatment. Much important information has been gained by his study of this series of cases, and I am sure many new and helpful ideas will be based on his conclusions. His remarks on the popularity of sinus disease are quite apropos, I think, but I am sure our conception of the subject is about to undergo a revision which will change our diagnoses, our treatment and our prognoses in many instances.

As the result of information gained within the past few years, my ideas concerning the cause, the treatment and the prognosis of sinus disease have been revised to a great extent. Formerly we were taught and we believed that practically all sinus disease was caused by infection, and we could not understand why ventilation and drainage did not always secure the desired results.

A chronic nasal discharge with frequent and persistent head colds and headache plus hazy X-ray plates were thought to be sufficient evidence upon which to base a diagnosis of chronic infectious sinusitis. Time and again have I heard the X-ray man, the medical man or others make a positive diagnosis of sinusitis on the X-ray evidence plus the history.

Since the rhinologic history and findings, including the X-ray plates in allergic individuals, are so similar to those seen in chronic infectious sinusitis, and since these individuals are so numerous, it is of first importance that we differentiate the two conditions.

The necessity for such differentiation is further emphasized when we realize that the nasal mucosa of allergic individuals is apparently less resistant to infection, that a very large percentage respond in a satisfactory manner to anti-allergic treatment and that only a small percentage require surgical interference.

It is my feeling that no case of chronic sinusitis should be put up for operation before having been studied for allergic reaction.

If this step is carried out I am certain the number of sinus operations will decrease very greatly and the number of cases improved as well as those relieved will be materially increased.

The etiologic relationship of sinusitis to asthma, it seems to me, is involved with the question of bacterial allergy, and it may be true that some of the good results which follow operations on the sinuses as well as those which follow tonsillectomy are based on a similar relationship.

It is not clear to me whether Dr. Richards has treated his asthmatics as allergic individuals or whether he has considered their sinus lesions as purely infectious in nature.

A classification of sinusitis as either infectious sinusitis or allergic sinusitis might convey what I am trying to get at—of course, keeping in mind that an allergic individual may contract an acute infection or he may have a chronic infection which will require surgical drainage.

While the essayist has not referred at length to the subject of headache, I would like to add that I have seen relief afforded many times for the most persistent cases of headache by anti-allergic measures.

In conclusion, it seems to me the working prognosis depends primarily on the cause of the disease.

Dr. R. G. Means, Columbus, Ohio: I want to compliment Dr. Richards on this paper because, after quoting statistics, he leaves the matter wide open and physicians can draw their own conclusions as to treatment, either medical or surgical. It, of course, is dangerous and Dr. Richards may find his figures twisted to meet someone's needs. The surgical cases always need medical consultation, both before and after operation, and in some of those treated medically it is doubtful whether they should be treated entirely from this standpoint or whether they should not also have the advice of the rhinologist.

A year ago I compiled statistics on 100 such cases as Dr. Richards has shown, and as he has told you, it was very difficult to prepare and properly classify the varying symptomatology. Likewise, the replies to follow-up letters are often difficult to analyze and put into figures. I believe the allergic cases in my series ran about 12 to 15 per cent. Many of these children had asthma, and a number of these asthma cases were treated surgically due to manifest sinusitis. I think when we speak of allergy it is hard to define it, as it is so often accompanied by infection and the sinuses are so frequently the foci that they must not be overlooked. I believe the bacterial and allergic types occur together in asthma more often than does the allergic type alone.

In the chronic group, speaking of the so-called "cures," it is doubtful whether they ever recover completely because so many have a persistent cough and many have bronchorrhea. Of course, the primary focus now

may be changed to the chest. Even though their symptoms are relieved, they are subject to recurrent exacerbations. The cases which are treated conservatively—that is, by washing out an antrum, or sometimes by doing a tonsillectomy, suffice to clear up many; whereas, others require procedures of a more radical nature. Many of those who had intrameatal openings in my series gave a history of marked improvement; in others, the symptoms were merely alleviated. I think in a definite small group who are not relieved by more or less minor procedures, we should be very radical in order to forestall devastating complications.

It is a courageous study that Dr. Richards has undertaken, and we hope he follows up these cases, aiding us in classifying them, and will also discuss the treatment of these youngsters who depend so greatly on us for their guidance and ultimate good health.

Dr. Lyman G. Richards, closing: I had no intention that this thing should develop into an intersectional contest. I am pleased that we have been able to demonstrate that, in spite of the fact that it took ten years to do it, we have at last learned from our St. Louis friends that there is such a thing as sinusitis, and have perhaps even demonstrated that we have learned in a way how to handle it.

I think Dr. Proetz is right; that we must not take these statistics as indicating anything final. I did not give them today with that idea; nor were they given to prove the excellency of any one form of treatment. The 60 per cent cures are by no means the figures you will get from any one treatment. We got these figures together to see how these cases acted after ten years. We had a record of the treatment in a given case that we considered was the answer to his problem. Much of the improvement shown came from a change in habits and living conditions. These cases must be taken to a hospital and gone over, medically and surgically and in every other way, and it must be done from the individual standpoint, not from the standpoint that any one particular treatment is a cureall.

Dr. Proetz suggested that I take about 75 cases and study them more intensively and bring out more detailed data, such as, study of their diet and home conditions, and try to estimate the actual future of the group when dealt with more completely than these cases have been.

I think Dr. Alden is correct as to adenoidectomy versus tonsillectomy. It is hard to say, when we do both operations, which one is of the most value. We do an adenoidectomy, and then fearing that by leaving the tonsils we may leave a factor that will make trouble later on, we are prone to do the double operation in the hope that it will cover the ground. Possibly that may be a mistake.

I said nothing about asthmatic cases. I think probably 50 per cent of these chronic cases had asthma. We inherit these from the anaphylactic clinic. It is a hard problem—that of asthma and sinusitis—and we do not know the answer yet.

If you take away anything from this study, I hope it will be that these cases must be investigated as patients before they are investigated as cases of sinusitis.

MANAGEMENT OF THE FRACTURED NOSE.

E. Fulton Risdon, M. D., F. R. C. S. (Canada),

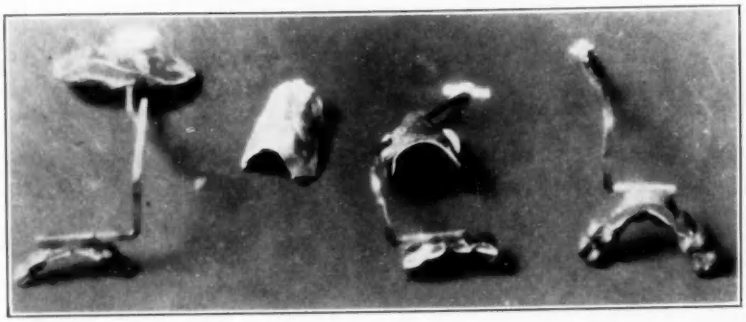
Toronto.

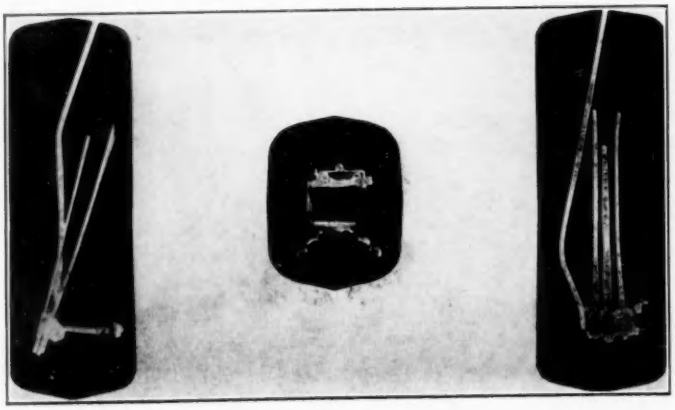
In considering this subject, we have divided it into recent fractures and old fractures with deformity. In the recent fracture the cause is usually sudden force, as in automobile or aeroplane accidents. The diagnosis, as a rule, is simple, due to the visible deformity, crepitation on digital examination, some nasal obstruction, and under most conditions certain X-ray evidence. We do not believe that X-ray is of very great value in the uncomplicated nasal fracture, as one usually arrives at a conclusion clinically, but the X-ray should always be taken from the medico-legal standpoint. The prognosis from the standpoint of deformity should always be guarded, but we believe if adequate splints are at hand very good results should be obtained.

The treatment is reduction of the fracture, or better, elevation of the fracture and immobilization of the parts. Of course, there are a number of these nasal fractures that do not require a definite form of immobilization but when they are comminuted and complicated, as one sees cases due to sudden violence, a splint is indicated. We have tried packing with gauze, and the use of porous internasal splints, which are merely left in the nose as packing, but we have been driven to the conclusion that the ideal splint must elevate the depressed and fractured nasal bones and spine, and at the same time mould the nose externally. We are now using a splint which we believe is most useful, in that it is fixed, the internal elevation necessary to obtain the correction is easily gained, and at the same time attached to this same splint is a bar which serves to maintain the nose externally in its proper relative position. It is generally necessary to maintain the splint in position for from five to eight days in the recent fracture, but when it is complicated by a fracture through the superior maxillary bones, then the splint maintaining the upper jaw in position should not be removed under five weeks. Further, where there has been laceration about the face, splints are still of considerable use, but, if it is possible, the lacerated area should be exposed,

as we strongly believe that seldom is a dressing necessary on the face, and when sutures are exposed to sun and air they remain dry and cause the minimum of scarring. Most of the sutures introduced about the face should be removed before the fourth day. We strongly approve of horsehair for superficial sutures.

We will attempt to show by lantern slides the types of splints we favor, but will here attempt a brief detailed description of such. The first type we wish to describe is one used when the nose has been fractured, perhaps laterally, but where there is little or no impaction or depression. In these cases we use a case appliance made to fit the upper teeth with a bar running at an angle to the forehead, and this bar attached to the forehead with a flat piece of dental compound, which when chilled will be held by adhesive. That means that we have an upright bar close to the nose on either the right or left side, whichever is desired, and fixed in two positions, namely, to the teeth and to the forehead. A piece of thin lead is now moulded to fit over the reduced or remodeled nasal bones, and this is held in position by further dental compound, which projects far enough laterally to engage the wire which we mentioned, fixed to the teeth and forehead. When this compound has been chilled, the nose is firmly held in the center line. So much for the fracture, which is not depressed, but most of the nasal fractures we see today are depressed, owing to the comminution of the nasal bones and the nasal spine. There is a lack of support when the parts are elevated, and we have tried packing with gauze, but gauze is likely to slip to a lower position in the nose unless the packing is very firm, but for fear of meningeal irritation we do not favor nasal packing. We have attempted to construct a splint which we think is more ideal than the one previously described, because it allows for elevation and at the same time pressure can be made over the nasal spine and nasal bones externally and internally. In this way we not only obtain elevation, but mould the bones externally in the upper nasal region. The appliance itself is similar to the one described, with the exception of a little attachment to the upright bar which permits a movable cylinder to lie at right angles to the nose, and this cylinder is held in position by a little lock or nut. To this movable cylinder is soldered three square tubes at right angles to the cylinder, and in these square tubes square wires are inserted, two of which wires are for extension into the nose and the third one, more centrally placed on the cylinder, for an





external wire to allow for pressure on the nose. The two inner square wires are used to elevate and the outer square wire to control the elevation, as it may be attached by dental compound, as we described before, to a moulded lead plate, overlying the nasal bones. This cylinder may be rotated on the little bar attachment and in this way give further elevation and then held in position by a small nut. In other words, we have this upright attachment which is fixed to the teeth and the forehead, to which is soldered a bar at right angles to the fixed wire and covering the little attachment is the cylinder which may be turned and held in position where the elevation may be needed, by a little nut. These square wires we spoke of passing through the small square tubes on the top of the cylinder and well up inside the nose, permit of elevation on rotation of the cylinder. It is difficult to describe any apparatus, but we will show it perhaps in a clearer way by the lantern slides.

In the old fracture, where the parts have been allowed to heal in a malposition, we refracture and attempt to bring the parts to their original position. We have tried using chisels and saws for refracturing, but we have found that most of the cases could be well handled with a large flat duck-billed forcep, intranasally. All incisions are made intranasally, and when the nasal bones and nasal spine have been refractured, it is necessary, as a rule, to hold them in an elevated position. Here we find that the movable splint described above is quite satisfactory. In many of these cases we believe that a total submucous resection is necessary, but there are cases where a submucous is contra-indicated, especially where, perhaps owing to an accident, there is a loss of nasal bridge support. At any rate, most cases requiring this form of treatment will be helped by a submucous resection, which is done at the same operation but previous to the refracturing. It has been our experience that a large percentage of the cases requiring refracturing of the nasal bones and the nasal spine will require a further operation two months later to supply the additional support in the form of cartilage. There is flatness about the old depressed nose which is not overcome by refracturing and elevation alone, but which requires a cartilage transplant to obtain a pleasing result. By that we mean a result may be obtained whereby a man may resume his employment without the stigma of syphilis. Many of these patients have been denied employment because of their deformity. We have used ivory and gutta

percha in place of cartilage, to obtain this elevation, and in some cases the ivory seemed quite successful. We have one or two cases where the ivory has been in for five years, but a large percentage of them returned with the ivory projecting either through the mucous membrane of the nose or through the columellar incision, and it had to be removed. Consequently, we believe that cartilage is preferable to bone or any artificial substitute.

This paper is written pleading for more attention to be given to the fractured nose. We believe that these cases are neglected, not because the deformity is overlooked, but due to the difficulty in obtaining adequate splints to immobilize the fractured nose. We do not know of any universal splint on the market that is of value, and consequently we have attempted to describe what we are using, hoping that many suggestions will be made for the modification and improvement of the present methods of nasal splinting.

DISCUSSION.

DR. T. E. CARMODY, Denver: I think we are to be congratulated on a paper of this kind by a man of Dr. Risdon's experience and on such an important subject.

His plan is very ingenious and his treatment of these cases is ideal if you could see him treat them. I am sorry we cannot see his slides because I know his work and think it is excellent. He has maligned himself in saying his results are not good because I can contradict that statement.

The Doctor speaks of accidents and makes a plea for better treatment. This should be presented to the general practitioners, the emergency surgeons and the general surgeons, because these cases come to us about two or three weeks after the accident and it is too late—there is usually a great deal of infection and we cannot treat the cases as we would like. If we can get these cases immediately after the accident, or within 24 to 48 hours, there is a possibility of doing something for them.

The cases that are difficult to treat are those where the whole face is mutilated, where there is not only a fracture of the nasal bone but of the frontal sinus, the superior maxillary, and sometimes the mandible. In those cases we have no fulcrum from which to make leverage; splints do no good; packing with gauze does no good, especially if the case is allowed to go for several days or a week before we see it. Fortunately, we do not take care of patients in accidents from aeroplanes. Usually the undertaker does that.

I agree with Dr. Risdon thoroughly as to the suturing of the skin. These sutures, he says, he leaves in four days. I go him twenty-four hours better. I usually remove the sutures in seventy-two hours. Some very fine suture is the one of choice, and we leave the wound without any dressing, and usually have very good results.

With the depression of the nasal bones, if we have no depression of the superior maxillary, Dr. Risdon's splint is ideal. I have used the Bernays-Simpson splint to support the nasal bone for twenty-four to forty-eight hours, and after these splints we are careful to use no packing because of the possibility of damming back infection in the ethmoid region.

The Doctor speaks of refracture. It is a question whether to refract or do a submucous resection. In talking to him about it we came to the conclusion that it is a great responsibility to decide when to do a submucous resection in these cases and when not, and as a general thing we guess wrong.

Dr. Risdon speaks of two months, if I understood him correctly, following refracture for building up the bone with cartilage in cases where we have deformity. I think that is a question for the individual case, but I would say two to six months.

There is little to add since the Doctor covered the subject so thoroughly.

Dr. E. Fulton Risdon, closing: Just one point. The reason I said two months is that I feel there is a certain amount of residual infection following a refracture of the nose. I really meant to say three months. If one is going to replace the lost nasal support with cartilage it is pretty serious, because your patient will have two or three days of intense pain, and he will only allow you the opportunity of doing it once. We should wait until much of the residual infection has cleared up following refracture, and then the transplant can be used.

NASAL OBSTRUCTION AND SYSTEMIC CONSEQUENCE.

JOHN J. SHEA, M. A., M. D.,

MEMPHIS, TENN.

Nasal obstruction may be partial or complete, developmental or the results of trauma. The systemic consequence of nasal obstruction depends on the age at which the obstruction occurred, modified by the individual characteristics. The correction of nasal obstructions constitutes more than 60 per cent of the nasal surgery in our hospitals. Too often a rhinologist unconsciously asks himself during a nasal examination, "What do I see that I can correct?" His eye may discover a deflected septum or a hypertrophied turbinate, and immediately that bit of pathology is considered the cause of the patient's complaint.

Early obstruction tends to retard the pneumatization of the sinuses and the normal growth of the alæ nasi and upper maxilla. During adolescence the turbinates should develop, and any intranasal obstruction will retard their growth, and also cause frequent colds and headaches. The reflexes, the result of adult obstruction, are manifested as headache, neuralgia, vasomotor rhinitis, asthma and emphysema. The lack of oxygen is manifested systemically by weakness, anemia and slow cerebration.

DEVELOPMENT.

The paranasal sinuses can pneumatize normally with a complete occlusion of the vestibules or the choanæ;[1] on the other hand an early septal deflection which jams the middle turbinate against the lateral nasal wall may obstruct the infundibulum, blocking the nasofrontal duct or the ostium of the antrum, thereby arresting pneumatization of the sinuses which have their origin from this region.[2]

Case 1.—M. C., age 28, sustained an injury to the nose at an early age with a resulting deflection to the right. A roentgenogram showed on the right side a persistent infantile ethmoid and a rudimentary frontal sinus, while the cells of the left side were developed. The membrane of the right ethmoidal group was atrophic, in contrast to the hypertrophic membrane of the

left group. If the obstruction interferes with the pneumatization of the antrum, it will also affect the size of the palate and the position of the molar teeth. The buds of these teeth are in relation with the posterior wall of the antrum, and their eruption depends upon the posterior elongation of their sinus.

INCIDENT.

Deviated septa occur more frequently in civilized races than among savages.[3] Schaeffer states: "The nasal septum is . . . . encroached upon more and more by the forward cranial extension incident to brain growth as one passes from the anthropoids to the ultracivilized races of man." The same author has seen several markedly deviated septa in fetuses.[4]

The facial trauma, incident to a difficult birth, may dislocate the septal cartilage, and the future growth of this cartilage will produce a deflected septum and block the nasal passage. If the deflection is severely unilateral the turbinates will hypertrophy to fill in the concaved side, or the ethmoid cells of this side will overpneumatize and displace the middle turbinate mesially.

Trauma, the result of one single severe blow or multiple lesser injuries, will cauliflower a septum similar to a fighter's ear. The obstruction in these cases is high up between the anterior ends of the middle turbinates.

ETHMOTURBINAL OBSTRUCTION.

The adult ethmoid cell or turbinate may undergo a cystic degeneration with the production of a marked obstruction. It is this former class of obstruction which may cause direct pressure on the optic nerve.

Case 2.—N. L., age 39, referred by Dr. E. C. Ellett, May, 1921, with a history of failing vision in the left eye for eleven days, preceded by a week of headache over the same eye. The eye was sore at first but now was better. A general physical examination, including blood and spinal fluid Wassermann, had been done, as he had had a venereal sore in 1907 and its nature was uncertain. These tests were all negative. The vision was 20/20 R. and 1/200 L. The pupils were larger than usual, equal and active. The right eyeground was normal, but the left showed a swelling of the nerve 2 D. without hemorrhages or exudation. The left antrum transilluminated darker than the right but washed clear. The X-ray report was as follows: Septum deflected, frontals neg-

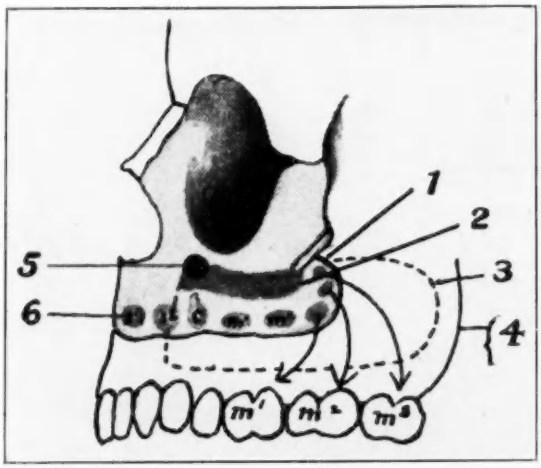


Fig. 1. The manner in which the development of the maxillary antrum affects the size of the palate and position of the molar teeth (After Keith).

ative, right ethmoids show increased density; the left shows a large cyst displacing its middle turbinates, and septum to the right. Thickened membrane in antra, more marked in the left.

The field was dull all over with a relative scotoma extending to 10°, and absolute central scotoma to the 3 circle. May 23rd, I operated on the sinuses. The left ethmoidal bulla was entered and the ethmoidal cells were found to have undergone cystic degeneration, the walls being like parchment. The left middle turbinate was removed and found atrophic from pressure. The posterior ethmoid cells extended internally to the sphenoid and obscured the ostium by being dilated in front of it. The septum was resected and replaced to midposition. No pus was present in any sinus. July 12th, vision, O. S., 20/20, but things looked foggy at a distance. The pupils were 5½ mm., equal and active. The left nerve was noticeably pale and the vessels were a little smaller than the right. The field was normal. A report three years later from this man was to the effect that he has had no further trouble.

HEADACHES.

The headaches which are caused by nasal obstruction vary from slight local nasal discomfort to severe neuralgias. The simple thickening of the septum high between the middle turbinates will

Fig. 2. Illustrating the manner of obstruction by
A—Accessory cell in middle turbinate.
B—Cell in uncinate process.

interfere with the normal physiologic swelling of these turbinates and produce the sensation of too tightly fitting nose glasses. These patients appreciate their obstruction when they remain out of doors during cold weather or during an acute cold. When they are well or in a normal climate their nasal passages are clear. This type of case receives a good result from a high submucous. A change of occupation may make a difference. A business executive, who formerly worked in the open, will be annoyed by a slight deflection due to indoor life with its lack of exercise. He may have had a marked deflection when he was outside, but the exercise and brisk atmosphere toned up his nasal circulation, thereby freeing his nasal air passages. In contrast to these simple high thickenings are the "orthopedic septums," with their marked deformities of the septal structures. These patients often suffer no discomfort or injury as the result of their obstruction, this being due to the compensatory atrophy of the obstruction on the lateral nasal wall. A submucous in these cases improves the looks of the internal nasal structure and provides against future trouble but may have no immediate benefit.

SYMPTOMS: THE RESULT OF THE SYMPATHETIC NERVOUS SYSTEM.

There is a deformity caused by a backward development of the sphenoidal process of the septal cartilage on a spur of the vomer. This large spur crowds the posterior fourth of the middle turbi-

Fig. 3.

High deflection of the septum. Orthopedic septum

nate and is capable of persistent trouble. Whenever the middle turbinates enlarge, contact will be made and pressure exerted directly upon Meckel's ganglion. This is very noticeable during the menstrual period. The growth of this spur interferes with the size and shape of the middle turbinate and by extending laterally will encroach upon the ostium of the antrum. This deformity favors maxillary sinusitis by blocking the ventilation and drainage of the antrum. Cocainization of the region of contact will temporarily relieve the headache of these patients.

Pressure on the nasopalatine ganglion may produce:[5]

1. Vasomotor rhinitis with rhinorrhea, sneezing, cavernous swelling, rosacea lachrymation, exophthalmos, mydriasis and photophobia.

2. Lower half headache with its pain behind the ear.

3. Ophthalmic migraine and the sympathetic syndrome.

4. Asthma. By a reflex through the nasal ganglion the sympathetic nerve supply of the bronchial tree can produce asthma. The mere presence of an obstruction in an asthmatic would not preclude that it was the cause of the asthma. Before the obstruction can be assumed as the cause it is necessary that the more usual causes, allergy, cardiovascular disease and nephritis, be excluded. It has been the experience of every matured rhinologist

to relieve an asthmatic by correcting his nasal obstruction, yet as commonly as this occurs, there are no physical signs or nasal symptoms pathognomonic enough that in a future case we can with certainty promise relief.

CEREBRATION.

"A good thinker needs a clear head." The dull child of hypertrophied adenoids is but an exaggeration of the adult with a blocked nose. The inability to breathe through the nose slows the cerebration and clouds the memory. As expressed in the words of Shakespeare, "A man so dull, so dead in looks, so woe begone."

GASTRO-INTESTINAL.

Whenever two surfaces of the nose, not meant to contact, are crowded together, they react on each other like a foreign body. If this contact is posteriorly, the excessive mucus produced will flow backwards. Some of this secretion is expectorated, but a quantity of it is swallowed. This latter portion enters the stomach and interferes with the normal gastric fluids. Whenever a non-ozena nose produces a bad breath it does so by this gastric disturbance.

EUSTACHIAN TUBE OBSTRUCTION.

A low ridge type of obstruction will enlarge the posterior end of the inferior turbinate and block the mouth of the eustachian tube. This same type of deflection is a danger to the ear, because it deflects currents of air or discharge towards the fossa of Rosenmüller whenever forced blowing of the nose is practiced. The correction of these low ridges facilitates inflation and often reduces the frequency of its demand.

INSUFFICIENT OXYGEN.

Oxygen is essential for the metabolism of the body, and when the obstruction is sufficient to reduce the minimum normal intake a temporary anemia is produced. This will demoralize the other functions and in time create a chronic anemia. Their cerebration will be retarded and their physical stamina diminished.

CONCLUSIONS.

The nose is wonderfully constructed and the maintenance of its free passages for ventilation and drainage is the rhinologist's contribution to mankind.

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DISCUSSION.

DR. G. F. HARKNESS, Davenport, Iowa: The systemic consequences of nasal obstruction *per se* that Dr. Shea has so ably and concisely presented merit careful consideration, for a survey of recent literature fails to find, with few exceptions, investigations that have confined themselves to this particular field.

Why do so many individuals with nasal passages that are markedly obstructive in type apparently suffer little systemic consequence? It is probable that it is because the phylogenetic development of the nose proves rather definitely, from embryological and comparative anatomical studies, that this passageway is primarily for olfactory purposes, and that secondarily it takes on a respiratory function in an adjunct capacity.

The development of certain well known physiological qualities on the part of the nasal membranes that pertain to the respiratory act behooves one to avoid needless sacrificing of mucosa. As St. Clair Thomson has stated, "It is better to be a partial mouth breather than to have free nasal passages with their protective mechanisms seriously damaged." Conservatism, however, must recognize that nasal obstructions can definitely produce a systemic effect.

Shurly, in 1927 (Arch. Otolaryn., March), quotes the experiments of Andersen of the Detroit College of Medicine who occluded the nostrils of guinea pigs, rabbits and puppies, with systemic effects varying from death to lowered resistance, predisposition to infection, impairment of vitality, dilation of the heart, marked changes in blood and skin, asthma and emphysema. Reopening of the nostrils gave relief from many of these conditions.

Anterior septal dislocations are to be considered purely from the standpoint of mechanical obstruction. The same applies largely to the occasional inferior turbinate which, from an overgrowth and bogginess of tissue, mechanically causes discomfort. It is variations in the upper and back part of the septum that demand the finer diagnostic discriminations. Here we have contact disturbances of tissues innervated from the nasal ganglion and with a most intimate autonomic nervous system association.

Repeated examinations with cocainization, limited to small areas of the upper and back of the nasal passages, will enable one to make a better differentiation as to nasal headaches and neuralgias.

Sir James Dundas Grant (Practitioner, 1929) quotes the work of Dixon and Brodie in 1903, who proved that stimulation of the nasal septum, especially in the upper and back part, can cause a loss of expansion of the lung due to contraction of the bronchial musculature. Sluder later gave us the results of his studies and the far reaching systemic effects

through the sympathetic nervous system in its association with Meckel's ganglion.

Infection is not pertinent to the subject at hand except insofar as the anatomical malformations of the parts are predisposed to infection. Suffice to say, the elimination of such a predisposing factor, when practical, may be the means of giving nature's forces the opportunity of overcoming the infection and should always be given consideration.

The author has stated that whenever a non-ozena nose produces a bad breath it does so because excessive mucus has entered the stomach and interfered with normal gastric fluids. This much advertised condition may come from dental disease, from the naso-pharyngeal tissues, from broncho-pulmonary disease, from some foods and condiments and certain metabolic and febrile disturbances; but how can mucus passing into the stomach so disturb the gastric secretions as to be an ordinary cause for halitosis? Normally air does not pass upward through the collapsed oesophagus. Prinz, in the Dental Cosmos, reports the experiment of filling gelatine capsules with finely chopped onion and feeding them to an individual, after which there was no onion odor to the breath.

Costellani, in fetor oris of tonsillar origin, has isolated two constantly present bacilli with the proposed names of B. colofoetidus and B. alkolofoetidus. On an agar medium the characteristic odor was obtained from the growth. I believe the gastro-intestinal factor is largely overrated.

I thank Dr. Shea for the privilege of discussing his excellent paper.

Dr. Fred J. Pratt, Minneapolis, Minnesota: I think this is a very important paper, because, as a rule, few men consider the general condition of the patient who presents himself with nasal obstruction. I believe a deflected and thickened septum is the underlying cause of nasal conditions. If the deflection occurs early in life, then the ethmoid cells on the convex side of the septum do not grow, they remain small; on the concave side these cells enlarge and fill the space. The mucous membrane on the convex side is thin and on the concave side is thick. In neither of these is the membrane normal; there is compensatory atrophy on one side and compensatory hypertrophy on the other. After a submucous resection, as the air passes through the nose, something happens to it and the general condition of the patient is changed. Watch a child before and after a tonsil and adenoid operation in which nothing else is done and notice the change in its general condition.

Dr. A. M. Alden, St. Louis: I wish to call attention to one factor in connection with this subject.

Many years ago, H. Lambert Lack, an English rhinologist, studied the mechanism involved in the development of the face, particularly the upper jaw. He found that the position of the tongue which normally at rest is in contact with the roof of the mouth, plays a most important part, in that the pressure of the tongue against the roof of the mouth produces certain constant lines of force upward and outward in both lateral directions, but which are, in turn, balanced by lines of pressure inward from each side by the movements of the muscles of mastication and expression. If nothing interferes to disturb this proper relation, the alveolar process,

palate, and the upper jaw develop normally and in proper symmetry with the rest of the face.

In the child, however, who, as a result of nasal obstruction, from whatever cause, becomes a mouth-breather, the normal lines of pressure exerted by the tongue are abolished and the inward pressure upon the jaw, exerted by the muscles of the face, remain unopposed. This results in the narrowing of the jaw with the formation of the so-called high arched palate. This is usually associated with abnormal prominence of the two upper central incisors. Inasmuch as the tissues making up the roof of the mouth form also the floor of the nose, when this floor of the nose is pushed inward, carrying with it, of course, the vomer, the cartilaginous portion of the septum which is fixed between the vomer and the perpendicular plate of the ethmoid, becomes buckled and gives rise to many of the so-called developmental deviations of the septum.

(Lantern slides illustrating the above condition.)

DR. F. E. HASTY, Nashville, Tenn.: Dr. Shea has very wisely laid emphasis upon the age at which obstruction occurs. I should like particularly to discuss nasal obstruction occurring in early life because it is in this group that obstruction is likely to do the greatest amount of damage. In fact, the possibilities of damage as a result of nasal obstruction in early childhood are almost unlimited. Not only may the adjacent structures fail to undergo proper development, but the whole body fails to develop properly when nasal obstruction interferes with aeration and oxidization within the body.

I have had some very happy results in children who have been properly cared for by competent orthodontists. By a certain amount of co-operation between the orthodontist and the rhinologist most cases of nasal obstruction in childhood can be corrected without any surgical interference. It is very important that the orthodontist thoroughly understand that as far as the nose is concerned the main objective is to spread the arch. This will require a certain amount of time, but very definite results can be observed within a few weeks after the beginning of treatment in children under ten years of age. It is my opinion that paranasal sinus infection in children is due primarily to nasal obstruction. This obstruction may be due to improper nutrition which is producing a congestion of membrane, or it may be due to improper development of the bony structure of the face. This, too, likely has for its basis improper nutrition and hereditary tendencies. I have seen at least three cases of asthma in children relieved by opening the nose in correcting mal-development of the arch. It is usually possible to give from one-eighth to one-quarter inch more space in each nasal passage.

Unfortunately, orthodontia is a comparatively new specialty, and not a sufficient number of men have been competently trained to take care of all work that should be done in this field. For people of limited means this kind of service is usually expensive. However, I have found it possible to get the orthodontist to co-operate in caring for my patients regardless of their financial means.

I should like to state that the results obtained from this way of dealing with children has really been a great joy to me. I feel that it puts

rhinology on a higher plane in that while we are relieving nasal obstruction, we do much for the general wellbeing of the child. I should like to urge my fellow-practitioners to consult orthodontists in their respective communities and to work and study with them for a sufficient length of time really to learn something about these cases before serious damage has occurred.

Dr. H. H. Vail, Cincinnati: There are several things about Dr. Shea's paper that I cannot reconcile with his well-known careful and accurate methods. One lantern slide showed a nasal septum which had two vomers, one at the top and one at the bottom.

Then in regard to the pressure upon Meckel's ganglion by a spur on the septum, I wish he would show how that happens. If we knew a little bit more about the infection of the nasal sinuses—how they begin, we could tackle this problem better. If I had my choice between a deviated antrum to operate and a deviated septum, I would take the antrum every time. I have seen a perfect septum operation, but the patient still has nasal obstruction from an antrum or ethmoid infection.

Dr. John F. Barnhill, Indianapolis: Dr. Alden's pictures are instructive, but unfortunately his descriptions of them were not full enough. Several years ago we proved that the normal pressure of the infant tongue upon the infant palate is necessary to normal development of the hard palate, and indeed of the upper jaw. If there is obstruction in the nose or nasopharynx of the child during the developmental period, the mouth must remain open and pressure of the tongue is then impossible. Faulty breathing, the open mouth, and failure of the tongue to exert normal pressure, are essential causes of palatal deformity.

THE DIAGNOSIS AND CONSERVATIVE TREATMENT OF SPHENOID SUPPURATION.

W. A. Wagner, M. D.,

New Orleans.

Excellent but few contributions have been made to the literature on the subject of sphenoid suppuration, and comparatively little original work has been done during the past several years. The sphenoid sinus, as is well known, is obscurely located, and although it is only a small part of the respiratory mechanism it nevertheless merits special attention.[1] Suppuration may involve the sphenoid separately, but is usually associated with an ethmoid involvement. Infection involving this sinus, with or without posterior ethmoid involvement, is most difficult and one of the rarely diagnosed of sinus infections.[2] The reason for this is apparent when one considers the mild and ofttimes vague symptom complexes together with the almost inaccessible location of these cavities. No doubt some sphenoids with a low grade infection have escaped detection for years, having been considered merely a chronic catarrhal condition, until the sudden appearance of a severe ocular disturbance or another complication invited immediate attention.

Of all the sinus suppurations, the sphenoid is probably most dangerous, because of its being surrounded by the meninges, the cavernous sinus and its contents, the carotid artery, the hypophysis and the optic nerve and chiasm. Therefore, to avoid any serious complications from extension of infections and to expect rapid recovery, early diagnosis and proper treatment are imperative.

A. DIAGNOSIS.

It is only by intelligent interpretation of subjective symptoms and objective physical findings that diagnosis reaches a firm foundation, and especially is this exemplified in the diagnosis of sphenoiditis. Sphenoidal suppuration, with which this subject deals, is less difficult to diagnose than the hyperplastic type, although "there is one clue," as Skillern[3] has said, and that is the "appearance of the middle turbinate." The body of this structure

appears slightly hypertrophied and frequently has a mottled appearance. Today one has learned there is more to the diagnosis of sphenoid suppuration than the mere appearance of the middle turbinate. The diagnosis is dependent upon a scrupulously detailed history and a carefully scrutinized study of the objective physical findings, the radiographs and the cytologic washings.

I. HISTORY.

The various symptoms associated with sphenoid suppuration are numerous and vary from simple headaches to the peculiar symptom complexes, some of which are seemingly unattached to the sphenoid. No doubt the morphologic variations are a factor in the symptomatology of this disease.[4] So evident is this that occasionally sphenoidal suppuration may simulate intracranial lesions, and because of the seriousness and magnitude of such an operation as an exploratory craniotomy, and the dangers of a sphenoidectomy, every procedure available should be used in the differential diagnosis.[5] Sphenoidal infection may occur in children, as Myerson has reported a death in a child, five years old, from meningitis in which the sphenoid sinus showed pus.[6] Shaeffer, Shea[7 8] and others have called our attention to the development of the sphenoid in children as young as three years of age, consequently a sphenoidal suppuration at that age is not impossible, and therefore one should bear in mind the possibility of a sphenoid involvement in childhood. Three such cases have come under my observation recently, corroborated by radiographs and cytologic lavage. MacGibbon, in a paper on spheno-ethmoiditis, states that this condition is common in sections of Australia, stating that 50 per cent of the children of Christ Church have infection of the ethmoid and sphenoid sinuses.[9] Although I do not doubt this statement, I do believe that we have less involvement of the sphenoid in this country. Children with impaired vision, as existed in my cases, in whom there is no pathology within the eye to account for the diminished visual acuity, should require an investigation of the sinuses, especially the sphenoid. Often we are surprised at the size and degree of development of this sinus in children. Radiography in the Hirtz position and cadaveric study have demonstrated this repeatedly.

Sphenoid suppuration may be acute or chronic. Acute suppuration manifests itself clinically with symptoms suggestive of

a cold, very closely resembling other acute sinus infections. It may be the initial involvement or it may be an exacerbation of a chronic one. The symptoms are very similar to those of the chronic except that the attack is sudden, the headache, neuralgic pains and tightness in the head are most intense and the vertigo may be pronounced. The optic neuritis may result in a transitory blindness and the field of vision and color acuity may be so affected as to produce a temporary color blindness and contracted visual fields.

The history is one of upper respiratory affection, with a tendency to recurrent head colds, headaches; throat, nasal and mental symptoms, ear and ocular manifestations, and many general symptoms apparently referable to the sphenoid.

Headaches are produced as a result of toxemia and pressure within the sinus, rarefaction from vacuum, or irritation of the Vidian nerve and the sphenopalatine ganglion resulting in the "front half" type of neuralgic headache in which the pains are typically sensory along the distribution of the fifth cranial nerve, and the "back half" type myalgic with myalgic nodes, vasomotor in origin, along the distribution of the occipital nerves, accounting for the Vidian syndrome and Sluder's "lower half headache."[10] Pain originating in the eye may be found radiating to the temple, to the mastoid region and to the nape of the neck.[11] The mastoid pain may become such as to simulate the pain of mastoiditis. Firrot, in 1927, cited three cases of simulated mastoiditis in which the patients were relieved by sphenoid drainage.[12] Occipital pain, so frequently a manifestation of sphenoid infection, is the result of Vidian nerve irritation in which the reflex is carried through the great deep petrosal nerve to the carotid plexus, thence to the superior cervical sympathetic ganglion, to the cervical plexus, and finally through the three upper cervical spinal (occipital) nerves. Occipital pain localized over the mastoid is often associated with vertigo and tinnitus, and when occurring together is spoken of by Oaks and Merrill as "the sphenoidal sinus syndrome."[13] Bertermes, in 1925, reported a case with occipital and mastoid pain in conjunction with wryneck, in which rapid disappearance of the pain and torticollis occurred after sphenoid treatment.[14] The sphenoid discomfort varies from a heavy, steady aching to lancinating, stabbing pain or a burning sensation; and the area described is often sensitive to superficial pressure.

Throat and nasal symptoms are: Coryza, as before mentioned, postnasal discharge occasionally with scab formation and constant hawking, often associated with hoarseness or aphonia; anosmia; subjective odor; parosmia; dry cough; clearing of throat and gagging.

Mental symptoms simulate those of neurasthenia and may be severe enough to produce delirium.[15] They may be best expressed as Citelli's syndrome, which was again called to our attention by Caleciti in 1925, characterized by defective memory, somnolence, insomnia, intellectual deficiency and unwillingness to concentrate.[16]

Ear symptoms are: Diminished hearing, tinnitus, vertigo, fullness, otalgia and splashing in ears. Sluder,[17] 1926, called our attention to diminished hearing with an associated fullness, and attributed it to eustachian salpingitis resulting in catarrhal deafness. During the same year the superior cervical sympathetic ganglion syndrome, described by Palmer,[18] gave an explanation to account for the tinnitus and vertigo which so frequently accompanied sphenoid infection. Through irritation of the superior cervical sympathetic ganglion which supplies the nervi vasorum of the internal auricular branch of the occipital and the auditory arteries to the labyrinth, hyperemia of the organ is produced, which brings about the tinnitus and vertigo. Therefore, vertigo and tinnitus are not due entirely to toxemia. Vertigo may be expressed as "giddiness" or "light-headedness," or a "feeling of falling." It is usually experienced upon motion of the head or when walking, stooping or turning.

Ocular manifestations are: Redness of eyes, pain in and about the eyes, asthenopia, muscle imbalance, photophobia, impaired vision from optic neuritis or atrophy, and always suggestive are the refractive states that vary with repeated examinations and little improvement. Diminished visual and color acuity and concentric contraction of the color fields often associated with central scotoma (relative or absolute), enlargement of the blind spot and pallor of disc.[19 20 21 22 23 24 25 26 27 28 29 30 31 32 33] One should be cautious in considering sphenoid infection as a cause of optic neuritis, as thirty-four other causes have been enumerated by Vail.[34]

(1) Multiple sclerosis, (2) acute myelitis, (3) Leber's hereditary optic atrophy, (4) tobacco-alcohol myopia, (5) diabetic amblyopia, (6) neuritis from a gravid uterus, (7) lactation, (8)

sinus infections, (9) infections and toxemia, (10) cerebrospinal meningitis, (11) tuberculosis, (12) typhus, (13) erysipelas, (14) beri-beri, (15) influenza, (16) mumps, (17) pneumonia, (18) angina, (19) meningitis, (20) encephalitis, (21) smallpox, (22) measles, (23) pertusis, (24) vaccination, (25) Bright's disease, (26) severe burns, (27) carcinoma, (28) tumors of frontal lobe, chiasm and posterior fossa, (29) alcohol (methyl), (30) tabes, (31) quinin, (32) lesions of third ventricle, (33) abscess of brain and (34) hyperthyroidism.

General symptoms are nausea, vomiting, gastric irritability, respiratory disturbances, arthritis, polyuria, polydipsia, afternoon fever and increased pulse rate.

II. OBJECTIVE PHYSICAL FINDINGS.

Because of the diversity and great variations of the subjective symptoms of suppuration of the sphenoid sinus, it must be remembered that a reliable diagnosis cannot be based upon symptoms alone, consequently objective physical findings play an important part in the diagnosis.

The rhinoscopic findings: There may be a septal deflection with secretions in the olfactory fissure noted after overcoming the turgescence. The pus appears either anteriorly between the middle turbinate and septum or posteriorly in the nasopharynx occasionally with crusting, and located toward the choana. Often the oropharynx shows a dry, glistening appearance, periodically streaked with pus, and while at times the secretion forms in crusts on the roof of the nasopharynx it appears as fluid pus in the nasal cavity. When the drainage of pus is scanty, nasal suction may facilitate its detection within the nose. In addition to the turgescence the inner side, and particularly the posterior tip of the middle turbinate, may show hypertrophy or hyperplasia, and if the case is of long standing the mucosa may assume a translucent appearance not unlike a beginning polyposis.

The presence of pus on the cotton of an applicator passed into the olfactory fissure or into the sphenoid sinus demands further investigation. Nasopharyngoscopy is of much assistance, although it may not always bring the ostium of the sphenoid into view, it frequently gives a fair view of the pus streaked olfactory fissure and spheno-ethmoid recess. It demonstrates hypertrophied turbinates with their polypoid posterior tips, dilated blood vessel, granulations and edema about the sphenoid ostium.

The pharynx, in addition to the periodic dryness and its pus streaked wall shows the typical granular and follicular pharyngitis with its enlarged or suppurating posterior pharyngeal lymph nodes and scar tissue infiltration. The ears may reveal a catarrhal deafness, noted by the functional tests and findings in the middle ears.

III. RADIOGRAPHY.

The consensus of opinion is that radiographs of the sphenoid seldom show pathology. This opinion, no doubt, was brought about by the unsatisfactory radiography of the sphenoid sinus in the past, which led to erroneous conclusions. This was due not only to faulty technic and lack of standardization of positions but especially to the routine use of a single position. It is my practice to require two positions, the Granger,[35 36] 107° angle (anteroposterior) and the Hirtz chin-vertex (vertical),[37] supplemented by the Rhese (oblique)[38] and Scheier (lateral).[39] Radiopaque substances, such as lipiodol, are used as an auxiliary agent. It is of value in diagnosis of obscure conditions of the sphenoid, as it serves a twofold purpose. It aids in determining the thickness of the lining membrane, and in the prognosis, as it is known that a long elimination time is indicative of a badly functioning membrane. Radiopaque substances may be instilled into the sphenoid or may be introduced by the Proetz[40] displacement method.

The Granger position is the most generally used. In sphenoidal suppuration the "G" line becomes blurred or disappears.

The Hirtz position I have used some time as the position of choice for delineation of the sphenoid, particularly when a contrast medium is used. This position is also of value, as it demonstrates extension of infection from the sphenoid to the base of the skull, as shown by Pfahler.[41] With the head in the proper position the fluid level is clearly defined, and with the aid of contrast medium, by changing the position of the head, it is possible virtually to reconstruct the cavity to be studied by means of exposures made in different planes. Because of the necessity of having a fluid level in making the Hirtz position it becomes necessary to have the patient in the sitting position with the head extended backward about 60°. In this position a fluid level will be seen in the sphenoid sinus, as well demonstrated by Baum.[42] When suspicious of an optic nerve involvement and radiography

of the optic foramen is necessary, the Rhese position is of value, as the foramen are clearly demonstrated within the orbits and their size and relation to the sphenoid are noted.

In the interpretation of radiographs the most essential point is the quality of the film. It is necessary that the film be of less density and less extreme contrast, so that it may show the minute bone changes, the presence of finer changes in the soft tissue and the presence of fluid within the sphenoid.

IV. EXPLORATION.

Exploration or irrigation of the sphenoid sinus as a diagnostic aid has no equal in certainty and rapidity for determining the presence or absence of pus within the sinus. Irrigation with cytologic study during a remission is of much diagnostic and prognostic value. During acute infections and exacerbations of chronic infections, microscopy is unnecessary, as the pus is demonstrated in the washings.

In many cases irrigation is done with little difficulty when the olfactory fissure is of sufficient width to permit the passage of the canula or trocar, but, on the other hand, greater diagnostic difficulties are encountered in cases in which the olfactory fissure is narrow and the anterior wall of the sphenoid and the ostium are not visible or a posterior ethmoidal cell overlaps the sphenoid.

Such a procedure is not free of danger, as penetration into the cranial cavity or hemorrhage may occur. Myles, in 1882, made the statement that the sphenoid sinus was beyond the reach of human attack. Zuckerkandl first offered the suggestion that the sphenoid sinus could be penetrated by means of a trocar, and since then Schaeffer, who first investigated the sphenoid, was soon followed by P. Watson-Williams, Sieur, Jacobs, Menzel, Hagman, Lobell, Speilberg, Ingals and many others.

Investigation of the sphenoid sinus should be confined to cases which present signs and symptoms which may be directly referable to the sphenoid. There are three groups of cases that require such investigation. Group 1 comprises such as manifest local and constitutional symptoms which could be traced to the sphenoid, such as occipital headache, tinnitus, vertigo (sphenoid syndrome); pain back of the eyes or over the mastoid, mental symptoms, nasal obstruction, postnasal discharge, etc. Group 2, those patients who develop a sudden loss of vision with optic neuritis in which no

cause has been found. Group 3, patients suffering with intracranial complications, such as meningitis, brain abscess and general sepsis of undetermined origin.[43]

The cases which comprise Group 1 require differential diagnosis, as they may easily be confused with posterior ethmoid disease.

The demonstration of suppuration in the olfactory fissure is a finding which indicates a disease of the sphenoid but is no absolute proof of its existence, as the objective symptoms are also somewhat characteristic of suppuration of the posterior ethmoidal labyrinth. Therefore, the two conditions should be considered together in the diagnosis until one has definite proof that the origin of the secretion is from the sphenoid sinus, and until then it is impossible to establish the diagnosis with certainty. This proof consists (1) in the direct observation of the purulent drainage from the ostium of the sphenoid sinus, and (2) in the demonstration of secretion from the sphenoid sinus by means of irrigation.

After a suppuration of the sphenoid exists the question arises, Is this the only source of suppuration, or is the sphenoid disease combined with an involvement of the posterior ethmoid labyrinth?[44] After irrigation of a sphenoid sinus containing pus, the reappearance of pus in the olfactory fissure proves its origin in the ethmoid labyrinth. And the observation of pus draining from the ostia of the ethmoid by means of the nasopharyngoscope also proves a combined ethmoid infection.

Groups 2 and 3 comprise cases in which the optic nerve is involved or orbital or intracranial complications are impending. In such cases one must agree with Lynch, Beck, Hurd, Eagleton and others that conservative treatment gives nothing more than passing help.[45] [46]

B. CONSERVATIVE TREATMENT.

Conservatism, however, is an entirely relative term, and its implications vary at different times. In this discussion it implies any management of this disease save surgery upon the sphenoid or other sinuses. The conservative or nonsurgical treatment of sphenoid suppuration must be resorted to in properly selected cases for its successful application, and under no circumstances should anyone believe the nonsurgical methods a panacea, nor

attempt to make them replace appropriate surgery in cases frankly surgical. Sphenoid surgery will ever play an important place in the practice of rhinology, but as the nonsurgical methods are better understood they will be given more and more consideration and possibly in the future supplant some of the surgery now in vogue.[47]

Although conservative treatment comprises constitutional and local management, it would not be proper to discuss conservative treatment without at least mentioning prevention of the disease.

I. PROPHYLAXIS.

Today scientific medicine is more and more concerned with the consideration of the prevention of disease, consequently if the opportunity be given prevention of sphenoid infection should be practiced. The prophylaxis of sphenoid suppuration calls for (1) prophylaxis of acute upper respiratory infection; (2) general hygiene, with the prevention and correction of physical causes, as lack of exercise, improper clothing, chilling of body, loss of sleep, dieting, excessive perspiration with exposure, overheating, bathing, draughts, constipation, etc.; (3) nasal and oral hygiene: the proper education of children as to how to properly blow the nose and cleanse the postnasal space, to avoid nasal irrigations, to properly and regularly brush the teeth and cleanse the pharynx; (4) predisposing factors, such as allergy, mechanical nasal obstructions to ventilation and drainage, as septal deflections, neoplasms, hyperplastic turbinates and foreign bodies of the nose, should be properly handled; (5) diet should be regulated, especially in regard to vitamins A and D; (6) the endocrines should be studied and any dyscrasia corrected; (7) all local infectious causes should be properly cared for, such as diseased tonsils, adenoids, teeth and gums; (8) plenty of sunlight should be recommended; (9) extrinsic and intrinsic factors which may cause or perpetuate a respiratory infection, such as exposure to heat, cold and excessive humidity in house or office, and gasses and vapors produced by poor ventilation should be avoided.

II. GENERAL TREATMENT.

Fortunately these cases usually respond readily to general and local management. When the condition occurs as a complication of some systemic disease (e. g., influenza, scarlet fever, etc.), the

general treatment must obviously include the treatment of that disease. The marked tendency to recurrence, even after constructive surgery[48] (submucous resection, turbinectomy, infraction of turbinates, polypectomy, tonsillectomy, adenoidectomy and teeth extraction), seems to prove a constitutional basis in many cases. Internal or constitutional remedies should, therefore, play some part in their management. The treatment in acute cases should begin with rest in bed and a thorough cleansing of the alimentary tract. In the chronic cases presenting evidences of lowered vitality or resistance constructive tonics should be prescribed, and in the overfed, full-blooded or plethoric cases careful regulation of diet, restriction of alcoholic drinks and regulation of bowels should be advocated. In all cases, smoking should be discouraged. Suitable regular exercise should be recommended, diet should be regulated so that patient receives sufficient vitamins and plenty of fluids with alkalinization.[49 50] Atropin in small doses frequently administered by mouth is an aid in acute cases in checking glandular activity. Iodids, urotropin, salicylates and colloids have been of questionable value. Occasionally when sphenoid suppuration is a part of a pansinusitis, the result of a metabolic upset incident to heart, kidney or gastrointestinal diseases, it becomes necessary to treat these conditions. When the background is specific, such as lues, tuberculosis, diabetes, etc., insist upon specific treatment in conjunction with the other methods of therapy. Proteid therapy (milk, omnadin, aosan, caseosan, novoprotein, phylogetan, terpichin, caseal, lactosen, yatren, casein, atomasin, alfasol, tuberculin and typhoid bacillus), bacteriophage, vaccines and leucocytic extract (Hiss) have proven of little value, although Herelli[51] has reported good results with bacteriophage in other infectious diseases, and it would be interesting to know its results in sphenoid disease.

III. SYMPTOMATIC TREATMENT.

Symptomatic treatment consists in the relief of pain and headaches by means of anodynes, narcotics, analgesics, physiotherapy and electrotherapy. Cocainization of the sphenopalatine nerve or injection of the nasal ganglion is recommended in those cases where neuralgia is an aggravating symptom not relieved by anodynes and in patients who resent surgical intervention. The restoration of nasal ventilation and the favoring of the discharge of secretions will be discussed under local treatment.

IV. LOCAL TREATMENT.

In regard to local treatment, the re-establishment of drainage and ventilation is the objective.

1. *Shrinking Solutions.*—Cocain, metaphedrin, ephedrin, adrenalin, adrephin, etc., have been used. The use of these drugs for shrinking the turbinates is so generally known that extensive discussion would not add to their recognized merits.[52] They may be used in the anterior nares or when enlarged turbinates or deviated septums make it impossible for the solution to reach the diseased area, the oral or nasopharyngeal route suggested by Pressman[53] is preferable.

2. *Suction.*—It may be used in cleansing the meatuses of the nose in place of irrigation and for draining the sinus when the head is bent forward with the ostium dependent.

3. *Packs.*—The packing of the nose with various silver preparations (argyrol, silvol, collene, ichthyol and glycerin) and dyes, as recommended by Dowling and Skillern, has not given me the results that others have claimed for them.

4. *Lavage.*—Lavage or irrigation of the sphenoid, in addition to being of diagnostic importance, is of great therapeutic value. It is surprising how the removal of a small mass of pus by lavage will give marked relief of symptoms. Lavage may be done as Lobell[57] has described, by means of the direct (through ostium), or indirect (by puncturing its anterior wall with a needle and forcing the return flow through the ostium) method. An indirect puncture of the anterior wall made after radiographing the patient with the needle in place facilitates the procedure and makes it an accurate and safe one. It is the method of choice in cases where the ostia are small and displaced or the pus very thick and inspissated. Lavage at times is impossible because of nasal obstruction. Many canulæ and needles have been used for this purpose.[58, 59]

5. *Displacement Irrigation,* as recommended by Proetz,[60] is of some value. Many drugs have been used, but normal saline has been my choice. The important factor in this type of therapy is the dilution of the pus, which facilitates removal, although at times it is difficult or impossible to have the fluid enter the sinus.

6. *Diathermy.*—Submucous electrocoagulation (Beck)[61] of the turbinate overcomes the turgescence and shrinks the hypertrophied or hyperplastic turbinate.

7. *Radium* has been recommended as an adjunct in cases associated with ethmoid involvement where polyps may interfere with drainage and ventilation of the sphenoid.

8. *Ultra-violet irradiations* of the nasal ganglion have been advocated by Odeneal[62] for the relief of pain.

9. *Steam inhalations* often aid in shrinking the nose and are cooling and refreshing to the nasal cavity and throat, and give relief from tracheal irritation in acute cases.

10. *Heat* applied to the side and back of the neck has been recommended for the relief of occipital and mastoid pain.

11. *Intubation* with silk retention catheter was discarded because of its irritation to nasal mucosa.

12. *Powder insufflation* of iodin, boric acid, etc., is found to be irritating.

13. *Instillation* of drugs, such as antiseptics and ferments, have been discouraging, and the use of oils, jellies and ointments in the sinus or nasal cavity are not theoretically correct from a pharmacodynamic viewpoint.[63]

14. *Ionization* of the sphenoid was used in our clinic as early as 1925 and discarded; nevertheless, Campbell found it in some cases especially useful.[64, 65]

15. *Besredka antivirus*[66] has had very little trial, although in my limited experience it offers sufficient encouragement to warrant further investigation. Of eight cases of sphenoidal suppuration treated, six were treated by instillation and are free from pus and symptomatically well for a period of one to eleven months. The other two cases were treated by the Proetz displacement method, using the antivirus, and both are apparently well. None of the above eight cases required surgery.

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DISCUSSION.

Dr. George B. Collier, New Orleans: Dr. Wagner has given us a wonderful review of the literature, but when I got the program of the meeting I was a little surprised at the title of his paper, considering his wonderful surgical ability. He did not have time to give us much of this conservative treatment. A burned child dreads fire, and I for one am back to the conservative treatment of sinusitis. If my sphenoids were to be opened I would want every possible checkup done. All these patients should have plenty of sunshine and plenty of vitamines before the sphenoids are opened.

Dr. R. M. Armstrong, Lexington, Kentucky: I doubt very much if a sphenoid infection ever occurs primarily. It does occur in very rare instances secondary to an ethmoidal infection. Personally I have never been able to prove beyond a doubt that pus did exist in a sphenoid sinus, in spite of the very large group of symptoms which the essayist has called to our attention and the many methods of diagnosis.

I am glad to see the trend of thought along conservative treatment. In recent years my associate, the late Dr. J. A. Stucky, and myself have performed very few operations on the ethmoidal labyrinth or sphenoid sinuses. There are many preparations which can be used for shrinking nasal membranes but the one that has given us the best results is a combination of ephedrin inhalant and oil of metaphen, which I direct patients to instill into the nostrils three or four times daily, while in a recumbent position, and have them report to the office every third day for postnasal irrigation with a hypotonic solution. Following this the sphenopalatine ganglion is cocainized (4%) on the infected side and a firmly wound applicator is dipped in (6%) trichloracetic acid and placed in the olfactory fissure and gently passed up to or in sphenoid ostium if possible. This is allowed to remain in position for a few seconds and then removed, followed by application of (5%) mercurochrome. This treatment as I have outlined, together with supportive measures and regulated diet restricting carbohydrates and starchy foods, has given us excellent results.

SOME EXPERIMENTAL AND CLINICAL OBSERVATIONS ON THE "RETICULO-ENDOTHELIAL" COMPONENTS OF ACCESSORY SINUS MUCOSA.*

RALPH A. FENTON, M. D., AND OLOF LARSELL, Ph. D.,

PORTLAND, ORE.

Revaluation of numerous factors in the study of human immunity has made necessary the reconsideration of numerous data long accepted, among them a more accurate study of the minute structure of various tissues of the body. Difficulties of technic and in securing proper material have delayed such studies of sinus mucosa until very recent years. Not long since, for example, Mosher, Knowlton and MacGregor demonstrated the regrowth of ciliated epithelium after radical removal of sinus membranes.

The whole problem of the cellular content of normal and of pathologic mucosal surfaces has seemed to us of fundamental importance, in the light of late discoveries elsewhere. We have in other papers elaborated the analogies and theoretical considerations which started us upon the present series of preliminary experiments.[1] [2] It should here be stated that we have been unable to find any record of previous findings based upon so-called "vital staining" of the sinus membranes, except from some Italian observers who dropped the staining agent directly into the nose or throat.[1] We found that such direct application caused a diffuse connective tissue and nuclear staining, scarcely distinguishable from the use of ordinary general stains. No selective action was possible with such concentrations as 1 per cent trypan blue thus externally applied. We therefore resorted to extreme dilutions of trypan blue—one drop of 1 per cent solution to 15 cc. of normal saline solution, for surface staining of live human material in the incubator; and to the repeated injection subcutaneously of a few minims of the 1 per cent solution for selective staining of the tissues of live animals.

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Determination of the presence of "histiocytes," otherwise called resting wandering cells, clasmatocytes or "reticulo-endothelial" cells in sinus mucosa seems of considerable importance because of the marked phagocytic powers possessed by these cells, and because their presence in body tissues antedates the arrival both of lymphocytes and of polymorphonuclear leucocytes. The exact structure and behavior of these elements should perhaps first be defined; and the following summary is freely drawn from the last great work of Professor Maximow of Chicago, his monumental text on histology.[3]

The cellular elements of connective tissues are the following: (1) fibroblasts, (2) ameboid wandering cells, (3) undifferentiated mesenchymal cells, (4) mast cells, (5) resting wandering cells or "histiocytes." These histiocytes are numerous in richly vascular areas, but the number varies in different regions of the body—in general, they are about as numerous as the fibroblasts, among which they are scattered. Histiocytes are not ameboid; in form they are irregularly rounded or spindle shaped. The nucleus is smaller than that of the fibroblast and is oval or slightly reniform. The chromatin particles stain darker than those of fibroblasts and are coarser. The protoplasm takes a dark stain—in eosin-azur material a bluish gray tint—and has a distinct ragged outline. It is vacuolated, and living cells store trypan blue, lithium carmine, carbon particles, neutral red, etc., in these vacuoles. This capacity for dye storage is the most characteristic feature of histiocytes.

Various other terms, as clasmatocytes and resting wandering cells, have been applied to them, the latter because in inflammation these cells mobilize, become rounded and assume the röle of large ameboid, phagocytic, dye-storing elements.

Normally, transition forms between resting wandering cells and fibroblasts are rare, but in inflammation there are transitional stages. Under conditions of inflammation, the following three cell types appear: (1) leucocytes migrate out of the blood vessels at the beginning of the inflammatory process: they play their röle in the reaction and then degenerate; (2) fibroblasts are found: they divide mitotically, increasing in number; they form granulation tissue and later collagenous fibers. The fibroblasts are already highly differentiated and retain their original type. During mitosis, the processes are withdrawn, the cell body becomes rounded, but the daughter cells return to the usual

elongated form. Contrary to the teaching of some histologists, Maximow holds that fibroblasts never become leucocytes or wandering cells.

Endothelial cells also belong, with the fibroblasts, to the group of highly differentiated cells. In inflammation and congestive conditions they divide mitotically and appear swollen, but form no free cells and no granular cells. They appear, however, to form fibroblasts in late stages of inflammation. Miss Sabin, in 1923, held that they may be transformed into monocytes or histiocytes, according to whether they enter the blood stream or the connective tissue, and that they also may change from one into the other form. Maximow and many others do not accept this view. According to various authors, endothelium forms giant cells, polyblasts, phagocytes, etc., but these points are in controversy.

3. The third type of cell in inflammation is the mononuclear, which is nongranular, is phagocytic and has an oval or kidney-shaped nucleus. Under conditions of inflammation, lymphocytes and monocytes of the blood migrate into the tissues, undergo a rapid hypertrophy, and become transformed into large phagocytes, resembling the histiocytes already present. They become transformed into polyblasts, but can be distinguished easily from the latter by their smaller size. After two days they are all alike. Polyblasts of lymphocytic and monocytic origin also store vital dyes.

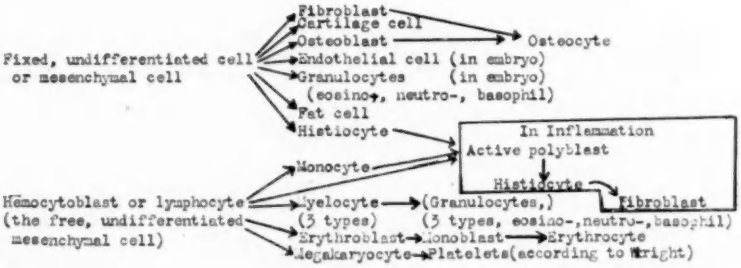
When local inflammatory stimuli act on histiocytes, they become free ameboid phagocytic polyblasts. Reticular cells of the lymphoid organs, spleen pulp, bone marrow, etc., undergo the same transformation. In the omentum this process goes on normally.

The polyblasts thus formed undergo various modifications. They may become: (1) epithelioid cells of the tubercle; (2) giant cells of foreign bodies, then fusion; (3) revert back to histiocytes, or (4) become fibroblasts.

In inflammation the histiocytes are mobilized in large numbers and become part of the polyblasts—i. e., the free, ameboid, phagocytic, dye-storing macrophages. Polyblasts thus have a double origin: (1) monocytes and lymphocytes of the blood stream; (2) histiocytes. But histiocytes arise in the embryo through direct transformation of lymphocyte-like primitive wandering cells.

Maximow and his school hold that the term reticulo-endothelial system is a misnomer, preferring the term histiocyte system, since they hold that the endothelium has no part in the activities of this system.

The so-called unitarian theory of blood and connective tissue formation is set up by Maximow in the following diagram:



Our objects in the study of experimental material were: (1) determination of the normal histiocyte content of sinus mucosa; (2) determination of changes due to (a) physical trauma (dull curettage); (b) physical trauma with heat (warm sterile paraffin); (c) bacterial inoculation; (d) physical trauma plus bacterial inoculation; (e) bacterial inoculation plus the subcutaneous injection of histiocyte extract (reticulin); (e) colloid injection (sterile 20 per cent gum acacia solution).

The frontal sinus of the cat was suggested because of its ready accessibility and relatively large size. (Fig. 1.) Under ether anesthesia a fold of skin is picked up and snipped vertically from the forehead above the inner canthus, leaving a wide "buttonhole" through which the periosteum is incised and separated laterally. The sinus is readily entered, either by a smart tap on a large antrum needle or by the use of an antral trephine. Through the small aperture made by the latter a dull ring curette readily may reach and traumatize the sinus mucosa. As the nasofrontal duct is relatively large and wide open, cotton may be packed into the sinus to retain solutions in place. Otherwise, with perfect drainage, the sinus wall rapidly cleanses itself. Paraffin proved an effective means for plugging the duct, and was used primarily to test the effect of an oily substance in histiocyte mobilization, in analogy to the employment of similar substances in the pleura and peritoneum for this purpose. Unfortunately, it had to be

rather warm to remain fluid in the needle, and the heat effects upon the mucosa will be described. Further experiment with other oils will be necessary before accurate conclusions are possible. Sterile gum acacia solution, 20 per cent, carefully filtered, was suggested by Dr. Ada Clark as a result of her work with Dr. Gay at Columbia on pneumotoxins in the circulating blood, and was expected to inhibit ciliary action.

The normal mucosa lining the frontal sinuses of cats bears few histiocytes or other elements of the so-called reticulo-endothelial system. (Fig. 2.) The histiocytes which are present tend to be distributed along the course of the blood vessels.

After mechanical traumatization of the mucosa there is a marked mobilization of histiocytes in the injured area and its environs. The submucosal blood vessels are congested and indications of inflammatory processes are numerous.

In sinuses injected with paraffin at a temperature somewhat above that of the body, considerable injury to the mucosa appears, due apparently to heat. (Fig. 3.) The epithelium is damaged and the subepithelial blood vessels are congested. Histiocytes are more numerous than in normal sinus membranes, but not so numerous as in the mucosa of sinuses traumatized by inoculation, which was done for comparison in the opposite sinus of the same animal. Besides histiocytes, numerous polymorphonuclear cells are found in the paraffin treated membranes—much more numerous than in those mechanically traumatized. Some of the polymorphonuclears are entering the epithelium. No eosinophiles—in slides differentially stained with eosin-azur—were seen in the connective tissue, although they are present in the blood vessels of the area.

Sinuses inoculated with a culture of hemolytic streptococcus (Fig. 4) show numerous histiocytes. The membranes are markedly swollen and congested. Many lymphocytes are also apparent. In animals allowed to live more than two days after inoculation there are also polyblasts and areas of fibroblasts, showing repair processes at work.

An animal inoculated with hemolytic streptococcus after traumatism of the sinus membrane, then injected subcutaneously with the histiocyte extract called "reticulin" (Charlton), showed a very large preponderance of polymorphonuclears in the membrane, which was fixed 24 hours after inoculation. The "reticu-

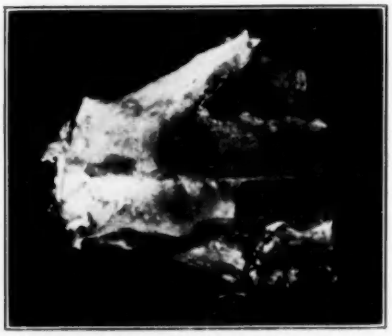
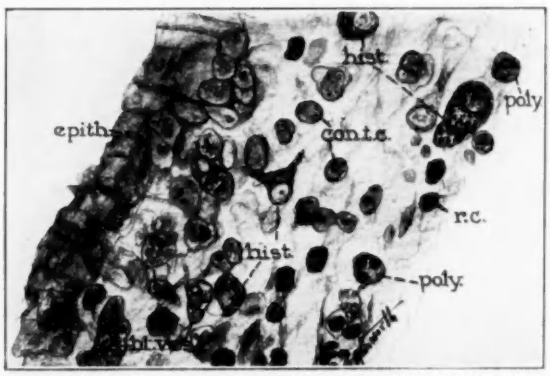


Fig. 1. Frontal sinuses of the cat. Fresh bone viewed from above after opening; fixed forty-eight hours after trypan blue injection. Membrane of left frontal sinus dark and thickened (below). Histiocytes have migrated into this membrane, which was traumatized. On the right side (above) membrane is clear and thin because histiocytes are very few under normal conditions. Natural size.



2. Normal mucosa and submucosal tissues from frontal sinus of cat, after vital staining by trypan blue subcutaneously. (Camera lucida stage 11, 1.8 mm. oil immersion with 10× ocular. bl-ves., blood vessel; con.t.c., connective tissue cell; epith., epithelial cell; go.v., goblet cell; hist., histiocyte; poly., polymorphonuclear; r. c., red cell; same captions and magnification in all figures.)

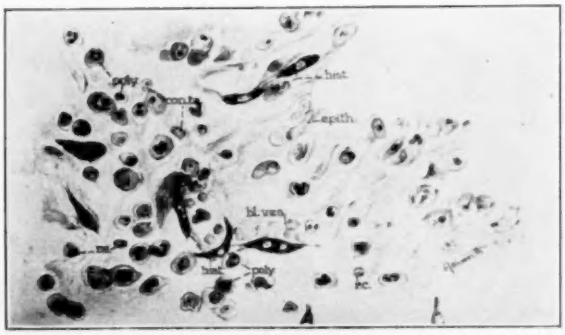


Fig. 2. Sinus membrane of cat following traumatism by paraffin above body temperarture.

lin" had apparently no influence in mobilizing more numerous histiocytes. Whether it was responsible for the unusual number of polymorphonuclears is not clear.

Inoculation of the hemolytic streptococcus into a sinus which had been curetted provoked a marked thickening and congestion of the mucosa (Fig. 5), with numerous histiocytes in the submucous area, and a few polymorphonuclears. Traumatism is thus seen to intensify the process which occurs after simple inoculation. (Fig. 4.)

Gum acacia solution, 20 per cent, caused disappearance of the cilia, with a slightly increased histiocyte migration and slight edema and congestion of the mucosa. (Fig. 6.)

These experimental animals were all killed and the material secured for preparation from 24 to 48 hours after the last vital staining injection had been made. In this manner late changes in the sinus walls, due to adventitious factors, superadded infections and circulatory differences were avoided.

Human material is obviously not obtainable by the method of vital staining through subcutaneous injection of dyes, because the color would appear in the sclera, skin and mucous membranes. Lithium carmine was tried but was found to stain human tissues so lightly in the doses used that the blocked histiocytes could not be identified readily; its color would be inconspicuous, and perhaps bolder experimenters may get some results with larger doses.

We therefore resorted to the staining of material immediately after its removal, from several cases of chronic maxillary sinusitis requiring the Caldwell-Luc operation. In most of these antrums lipiodol had been used anywhere from several days to a few weeks previously. All were highly edematous, polypoid membranes, some with small abscesses deep in the tissue, and all bathed in more or less foul mucopus. Good-sized pieces of this degenerated mucosa were carefully removed, placed instantly in 15 cc. of blood-warm normal saline solution containing one or two drops of 1 per cent trypan blue and then maintained at body temperature for two hours before fixation. Lithium carmine was tried in the same way but gave too light a stain for conclusive results. No delay or drying of these tissues was permitted, and in spite of the interrupted circulation a considerable penetration of the surface layers was secured.

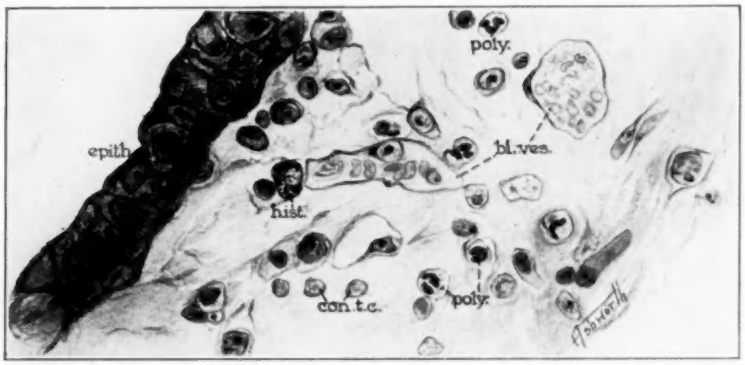


Fig. 4. Sinus membrane of cat following inoculation with hemolytic streptococcus.

Fig. 5. Sinus membrane of cat after traumatism by dull curette, followed by inoculation of hemolytic streptococcus.

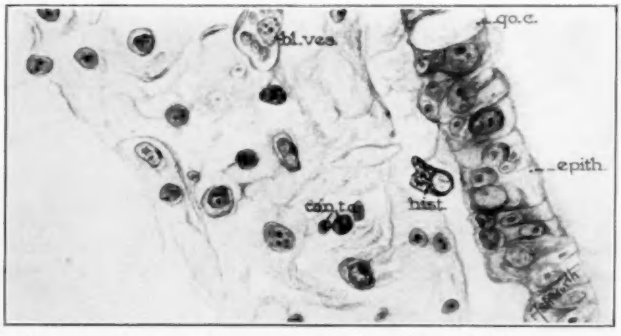


Fig. 6. Sinus membrane of cat after injection of sterile 20 percent solution of gum acacia.

No drawings of human material are shown, because of the relatively small series drawn from but one class of material. Complete study of human material would be outside the scope of this preliminary report, as it would require the differential staining of a long series, from the normal through the simple inflammations and allergic swellings to the various degenerative changes.

After fixation in 10 per cent formalin, sections of our human material showed fairly numerous histiocytes near the borders of the block of tissue and at points where the epithelium was broken. This indicated that the dye had not penetrated deeply enough into the block to be taken up by the histiocytes more deeply situated. It was therefore necessary to stain these histiocytes by some selective method. The eosin-azur stain of Maximow gave good results which could be checked. By this technic, the cytoplasm of the histiocytes takes a bluish gray tint and small vacuoles are well shown. The check on results consisted in comparing the cytoplasm of peripherally placed histiocytes which had taken up dye granules so that they could be readily identified, with that of the more deeply placed cells. Numerous cells of similar staining reaction, with vacuolated cytoplasm, irregular outlines and nucleus of round or oval form—in all respects save the presence of trypan blue like the peripheral histiocytes—were present in the deep submucosa. Lymphocytes were also numerous, and some eosinophiles were scattered here and there.

Professor Larsell's collaboration has been constant throughout this study, and he is entitled to the utmost appreciation for his friendly criticism and shrewd advice regarding the scope and the technic of this work in an uncharted field. His personal service was invaluable in the difficult task of microscopic preparation and study of a vast number of slides and in the choice of typical fields for illustration. Because of the relatively faint blue staining of the histiocyte and the necessity of a very light counterstain, microphotographs were not successful. We therefore owe much to the painstaking accuracy with which Miss Ashworth has made camera lucida drawings of the most important types.

This preliminary study is too brief to permit dogmatic conclusions; numerous animal experiments are required, with the use of differential and impregnation as well as vital stains.

It is, however, evident that in the cat, normal sinus mucosa, which is thin, ciliated and strongly resembles the human sinus

walls, contains histiocytes in small numbers near the blood vessels. Physical trauma and bacterial inoculation cause their arrival in considerable numbers. These cells are the first line of defense and repair, appearing ahead of the polymorphonuclears and the lymphocytes. Rapid mobilization of polymorphonuclears seems to be favored by heat changes induced by hot paraffin, as also by subcutaneous injection of "reticulin," a histiocyte extract. Gum acacia solution, a typical colloid, destroys ciliary action while slightly adding to the number of histiocytes.

Human material maintained in contact with a high dilution of trypan blue at body temperature in saline solution for two hours, after surgical removal from typical cases of chronic maxillary sinusitis, showed phagocytosis of the dye in the superficial layers. Rather numerous histiocytes were found throughout the tissue by differential staining.

Questions arising from the above fragmentary conclusions, which require further study and control under the most accurate laboratory conditions by physiologists, biochemists and pathologists, include the devising of methods for artificially increasing the number of histiocytes in the sinus membranes, and thus increasing the resistance and repair potentialities of these structures. This problem differs essentially from those of the peritoneum and pleura, heretofore thoroughly studied by Gay, Clark, Linton, Cannon and others. In the sinuses we have a ciliated, mucosal, air-circulating surface, instead of a serous one; and the sinuses are less resistant because of their vestigial character, while the pleura and peritoneum are physiologically very active. Because the sinuses tend by gravity and ciliary action to cleanse themselves, there is great difficulty in securing a continuous and total surface action from any local stimulant, such as broth, heavy oil, aleuronat starch or gum acacia. Perhaps the Proetz gravity and suction methods may prove of service in this respect.

Histologic investigation of the effects of various oily and aromatic substances, long employed empirically as inhalants, is suggested. Do these substances promote histiocyte activity? Further tissue work must also be done to decide how much difference in physiologic behavior and in histologic mutability may exist between the poorly aerated vestigial linings of the accessory sinuses and the highly vascular, well exposed surfaces of the turbinates and septum.

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DISCUSSION.

DR. FRANK J. NOVAK, JR., Chicago: I shall discuss the practical applications of the material which Dr. Fenton has presented.

If you take the trouble to search the literature you will find nothing bearing upon this particular piece of work which has been presented here today. If you take the trouble to study this paper carefully you will be impressed with the excellence of the work that has been done. It has the rare merit of being original, and unless I am greatly mistaken it will not have to be done again.

There is a great deal of confusion today as to what immune reactions really are. It was thought at one time that the changes which occur in the production of immunity are chemical changes in the fluids of the body, particularly in the blood. More recently, with the development of the new cytology and with the emphasis on the functions of the living rather than on the morphology of the dead cells, a newer conception has arisen, namely, that the mechanism of immunity lies in specific cells. We have, therefore, the humoral idea, and the cellular idea. And the most recent conception is that both the humoral factor and the cellular factor are concerned with the phenomena which we know as immune relations.

What Dr. Fenton has pointed out to us is something which was suspected and believed but which had not been demonstrated, namely, that in the sinus mucosa we have certain cells—as elsewhere, in the brain, liver and lungs—cells which are primarily concerned with the protection of the organisms against invading bacteria. He has shown, first, that the mucosal membrane has histiocytes, and he has shown that these histiocytes, the relative number and their concentration, can be influenced by a number of factors within the control of the individual; he has shown that some mechanical trauma, with a dull curette, for example, will increase the number of these elements. He has shown that heat applied to the sinuses will increase the number of these cells; he has shown that the injection of the sinuses with a virulent or massive dose of organisms will increase the number of these histiocytes. Further, he has shown that the injection of colloids has a direct influence upon the number found in the mucosa.

Now, to digress for a moment: I do not believe that there is a month of the year during which some antiseptic is not developed or proposed. I do not believe this screen is large enough to hold, in fine writing, the names of all the antiseptics on the market today. A part of yesterday afternoon was devoted to a discussion of the merits or demerits of a certain iodine and boric acid powder—"Does it work or does it not work?"

There has been a frantic quest for an ideal antiseptic, and I am sure the quest is just as futile as it is frantic. The practical implication of Dr. Fenton's paper is this: that the solution of the problem of protecting the individual—the sinuses—for instance—against pathological organisms does not call for the finding of an ideal antiseptic, but the finding of a method of controlling or influencing the immune mechanism which is already present. What are the means by which we can do this? Dr. Fenton has already pointed this out: Among other things, some chemical agents, heat or the injection of various antigers. It is not a question of what you think of the antivirus, or the saline suspension of bacteria, dead or living, or what other antigen is used. Certain fundamental knowledge is necessary before we can accomplish anything, and even as fine a piece of work as this is not sufficient. We have various ways of influencing the histiocytic system. We have the injection of dead organisms in a saline solution through the skin, or the injection of some protein material. We have the injection of sera, or sera plus organisms, dead or living, and we know the actual application, particularly of various antigens through the mucous membrane of the nose and throat, or skin, or into the skin; we know the effect these antigens have in controlling the concentration and actual number of histiocytes. Our problem, as far as I can see it, is largely this—the further determination not only of the histiocytes in the mucous membrane, but of other cellular elements that are concerned with immunity. Many cells, provided they have a large amount of cytoplasm, are active phagocytes. If they have a small amount they are not active phagocytes.

The problem then, as I see it, and the practical implication of Dr. Fenton's work is this: There are within the organism potential mechanisms for the protection of the individual against the invasion of pathologic organisms. The further investigation of these mechanisms will reveal the best means of influencing and controlling them.

Dr. Harris P. Mosher, Boston: If we are to make progress in dealing with infection of the mucous membrane, especially chronic progressive infection, before which we stand almost helpless, histology, pathology and biochemistry must give us new facts. The work done by Dr. Larsell and Dr. Fenton and just reported by Dr. Fenton, gives new information about the mucous membrane of the frontal sinus. Their findings are in line with those repeatedly made during the past eight or ten years in the examination of other body tissues, notably the peritoneum.

The reticulo-endothelial system is a very high-brow subject. Fenton takes to such a subject like a duck to water. For the rest of us it can hardly rank as human nature's daily food. The essayist's fundamental finding is that the mucous membrane of the frontal sinus of the cat responds to trauma and infection by an increase of histiocytes. This is the first tissue change. This finding is a step forward in our knowledge of the pathology of sinus mucous membrane. I hope that other constructive steps will soon follow.

With your permission, Mr. Chairman, as my contribution to this discussion, I should like to present six slides which show the part played by histiocytes in tissue cultures of the mucous membrane of a nasal polyp. We have obtained the same results in tissue cultures of antral mucous

membrane. Dr. P. J. Ross, working in my laboratory, is responsible for the observations which I now report.

Dr. Ross has succeeded in keeping tissue cultures of antral mucous membrane, and the mucous membrane of nasal polyps, alive for six days. He has made a distinct contribution to the technique of making tissue cultures by adding to his nutrient solution an extract of human umbilical cord. This is an easy way of getting an extract of embryonic tissue. The usual practice has been to use pig embryos. His results are summarized as follows:

Regardless of the pathological condition present, active cilia are found in all specimens. They retain movement en masse for 24 hours, and isolated cells show movement for 48 hours. The epithelium of the plant remains intact for 24 hours, then clumps of cells are pushed off by the growing cells. The dead cells retain their compressed form. This, of course, is most striking with the ciliated cells. The isolated live ciliated cells immediately become round. These round isolated ciliated cells Dr. Ross believes to be young, immature cells and the cells which take the active part in regeneration of the mucous membrane.

The first cells to come out—to speak colloquially—are the polymorphonuclears. On getting clear they show ameboid movements, and, of course, Brownian movement. They disintegrate in 24 hours, and in 48 hours are gone. The cells which predominate in the new growth and the cells which come out after the leukocytes and the large mononuclears, are the blood histiocytes. Almost at once they form a broad sheet at the edge of the plant and are free in the nutrient fluid. In 48 hours they lose their large mononuclear form and become branched. Fibroblasts, the near relatives of the mononuclears, appear at the end of 24 hours. Finally, at the end of 24 hours also, small lymphocytes appear.

Such are the results to date. The great difficulty in keeping the plant alive is contamination with the bacteria. Most of this comes from the pus present at the operation. Strangely enough, the preliminary washing of the plant renders it sterile in about one-third of cases. Few bacteria are found in the plant. This means, if it means anything, that they are killed by the protective juices of the cells.

In closing, I will quote from Dr. Ross one of his conclusions. It is startling in its present form, and if further work bears it out will be revolutionary.

"In approximately one-third of the cultures planted to date bacteria have been present which have overgrown the culture at the end of 24 hours. This percentage of infection has been less in cultures taken from nasal polypi than from antrum or frontal sinus membrane. It appears that the latter tissues are greatly traumatized and purulent material in the sinus itself is frequently and thoroughly mixed with the membrane during its removal. In the last series of 24 cultures, six cultures each were planted from four different specimens. Cultures from one specimen were bacteria free. In another, one showed bacteria. In the others, two and three respectively were infected. In the second, the organism was a streptococcus, in the others the organism was staphylococcus. In our opinion, the appearance of bacteria in these cultures is due to gross contamination dur-

ing removal and insufficient washing prior to planting on the cover slip. In view of the fact that the majority of the cultures are bacteria free, this conclusion appears to hold. In other words, actual bacterial invasion of the nasal mucous membrane prior to operation does not appear to occur from the evidence at hand, but the contrary. It is possible that bacteria in the tissues are dead, or there is sufficient local immunity present in the explant to prevent their growth."

Dr. Ralph A. Fenton, closing: I thank Dr. Novak for his fine statement of our problem, and also Dr. Mosher for the report of his marvelous work in tissue culture, which, by the way, is much more accurate than anything we can show on the animal, since it is subject absolutely to laboratory control at all times. It is beautiful work, and Dr. Ross stands alone in devoting attention to this part of the subject. I am most happy to have it brought up in connection with this paper of ours.

Increasing immunity by local means in these cases depends very largely upon arresting ciliary action (which has been so beautifully shown by Dr. Hilding at this meeting). This means that something has to be done to stop the cilia from working, and thus to keep the medicament in place long enough to soak in and do some good. Other measures which might be used are the action of heat, not directly but gradually, as by diathermy and deep X-ray. In other words, we must stir up phagocytosis. How does that happen in the normal? That is something about which we hope to hunt some facts to present to you at some other time. Then there is the question of the connection between the nervous system and the histiocytes. Why do these cells come in, under what influence, and where are the nerve fibers, the prolongation of the sympathetic nervous system, which bring about this action and which put these cells to work for us?

ATROPHIC RHINITIS.*

JOHN A. PRATT, M. D.,

MINNEAPOLIS.

In the latest ear, nose and throat book is stated that "Atrophic rhinitis is an atrophy of the nasal mucous membrane and the underlying soft tissues, eventually involving the turbinal bones. The etiology is unknown, as no pathologic condition or specific organism has ever been proven to be the causative factor." These same statements have been made in all papers and books written in the past to which I have had access.

Dr. Pollock, in his deductions from the paper of Dr. Vogel, and presented before the Middle Section of the American Laryngological, Rhinological and Otological Society, January 17, 1931, under the title of "A New Conception of the Etiology of Atrophic Rhinitis," states "The primary stage is a sinusitis (which exists in all atrophic cases), with subsequent infection and degeneration of the ganglion and thence leading to a similar degenerative change in its nerve fibers, which end in the nasal mucous membrane. As only a few atrophic cases result from nasal sinusitis, one must more or less assume the presence of predisposing factors, namely, hereditary tendencies and abnormal patulency of the nasal cavity." (ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, June, 1931.) This latest paper leaves us in the same position.

I beg to present the following hypothesis:

"That atrophic rhinitis is not a disease entity but a syndrome of hypoendocrinism, of which the adrenals play an active part, and is only one of many indications of hypoadrenia.

That it is not due to any anatomical condition, as an abnormally patulous nasal chamber. I have never seen a case of atrophic rhinitis where the turbinates have been removed or in negroes, who have very patent nasal cavities, but who are bountifully supplied by nature with adrenal products.

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That any nerve shock to the system, as infectious diseases, childbirth, etc., may so injure the adrenals as to reduce their secretory activity. The loss of the secretory stimulus or hormone on the vegetative sympathetic system may so weaken the heart muscle tone or blood vessel tone as to reduce the flow of blood to such terminal tissues as the skin, hair, mucous membrane and their underlying tissues. To compensate for this weakened terminal circulation, the terminal arteries and veins are reduced in size and atrophy is the result, or reduction of cell stimulus may bring the same end result of atrophy.

The mucous membrane of the nose is most noticeably affected, due to the reduction of the blood supply to the serous glands, causing an aqueous loss in the nasal secretion. The secretion must contain a certain percentage of fluid for physiological functioning of the nasal cavity. As the air passes through the nose, it takes up the aqueous portion but leaves the mucoid portion, thus one of the first nasal symptoms is the presence of a thick, tenacious secretion.

ETIOLOGY.

The underlying cause of atrophic rhinitis is a nutritive disturbance, due to an imbalance of the endocrine glands, particularly the adrenals. This condition is generally launched by a shock to the system, as infectious diseases, focal infections, changes from childhood to adolescence, pregnancy, childbirth, etc. If atrophic rhinitis is already present, any of the above named causes will increase its activity or may cure the case by re-establishing normal endocrine function. One frequently sees cases with atrophy of the turbinated bodies, but a functioning nasal membrane, where the crusts and discharge of early life can be elicited. These generally have compensatory growths in the region of the septum tubercle, uncinate process and the attachment of the middle turbinate. (See "Compensatory Nasal Growths," read before the North Dakota Academy of Otology, Rhinology and Laryngology, June 1, 1922, by the author.)

Atrophic rhinitis is never present in a hyperendocrine state, but only with a hypoendocrine function. The ozena is a sequela of the condition and is caused by the bacillus fetidus ozenæ or some other fetid germ. One investigator found ozena present 360 times in 1,142 cases of atrophic rhinitis—roughly estimated, about one out of three cases.

If there is excessive secretion, the attenuated mucous membrane of the fossæ and sinuses has been attacked by suppurating germs.

Suppuration and hyperplasia of the accessory sinuses in atrophic rhinitis are secondary and not primary, other than a focal infection.

The condition is not preceded by a hypertrophic rhinitis, and we believe that this theory is based on the fact that there is frequently a hyperplastic suppuration of the ethmoid capsule, which is caused by a secondary infection.

No type of skull seems to predispose to this condition, nor are the negroes, with their wide nasal fossæ, more prone to it.

Surgical removal of the turbinates causes dry nose but not atrophic rhinitis.

HISTOLOGICAL PATHOLOGY.

There is general atrophy of the mucous membrane and its underlying structures, even the turbinated bones. The ciliated epithelium, except islands here and there, is replaced by stratified squamous epithelium. The secretory cells of the mucous membrane of the nose are mixed, mucous and serous glands using a common duct. The glands are distinguished from each other by the fact that the serous glands have secretory capillaries, while the mucous glands do not. The connective tissue left in the underlying structure contracts and first obliterates the secretory capillaries of the serous glands and also interferes with the normal exosmosis of fluid through the membrane, thus taking away nearly all of the aqueous element of the normal nasal secretion.

This atrophy extends to the sinuses, pharynx, larynx and trachea, as well as to the normal lymphoid tissue. The atrophic degenerated state of the sinuses invites infection, thus producing a chronic suppurative sinusitis, accompanied by a purulent discharge.

DIAGNOSIS.

To successfully treat any condition, it must be diagnosed early. There are many signs and symptoms of atrophic rhinitis which appear long before we have the classic crusts, with their sequela of ozena.

The patient comes, either with reference to the crusts that form in the nose, causing obstruction, or because of the disagreeable odor complained of by the family or some friend. They are

often unable to discern this odor themselves, owing to the atrophic destruction of the olfactory nerve endings or from pressure of the hyperplastic ethmoid capsule.

The general appearance of the patient shows all the typical signs and symptoms of hypoadrenia: poorly nourished and anemic, with the senile appearance far beyond his years; thin, early graying hair, atrophic skin with its yellowish gray hue and premature wrinkles. They complain of fatigue and lack of vigor and are uninterested in the normal routine of life. The basal metabolism is reduced. The blood pressure is generally below 120. Pulse is slow and weak or rapid and thready.

Feeling along the side of the ridge of the nose where the lateral nasal cartilage joins the nasal bones, one will find atrophic depressions demonstrated by the sensation of touch, which in the later stages, are easily noticed by the eye (Pratt's sign).

On examining the nasal cavity the membrane will be found pale and the cavity larger than normal. There will be thick, viscid secretion clinging to the membrane and the patient may complain or at least confirm upon questioning that he has considerable viscid discharge which is hard to dislodge. This is due, undoubtedly, to the loss of the watery element of the secretion and the inability of the cilia to move it. There is not an increase of nasal discharge but rather the reverse, unless there has been a secondary infection of the sinuses. This change in the character of the secretion is noticed before any sinus changes by transillumination and X-ray can be demonstrated.

As the condition progresses, with atrophy of the membrane and enlargement of the nasal chamber, the loss of the aqueous portion of the secretion allows it to dry quickly by the passing air through the nose, and forms a crusty scale on the mucous membrane. In drying, the crusts pull away from the membrane, allowing the heavy mucus underneath to form a second crust. If the crusts are not removed they will eventually fill the nasal cavity. The action of putrefactive bacteria establishes the ozena. Keeping the nose free of crusts will banish the odor, unless the bacteria reach the sinuses. In atrophic rhinitis, when there is no odor, the crusts are dry. However, when odor is present, there is partial liquefaction and heavy mucus is attached to the crusts when they are removed.

The first perceptible structural atrophy usually affects the lower turbinate (due possibly to its blood supply coming from a ter-

minal blood vessel). A secondary infection may cause a hyperplastic ethmoiditis, which will fill the upper half of the nose, giving an atrophic lower turbinate and a seemingly hypertrophied middle turbinate. Polypi are rarely, if ever, present.

When the sinuses are transilluminated and X-rayed in advanced cases of atrophic rhinitis, they invariably show a sinusitis.

DIFFERENTIAL DIAGNOSIS.

With the symptoms, examination and findings as above, there will be no difficulty in diagnosing an atrophic rhinitis. A syphilitic condition can be differentiated by the ulceration and necrosis plus the Wassermann test. In cases of foreign bodies or rhinoliths, the odor is not present after their removal. Tuberculosis of the mucous membrane shows ulcerations. The sinus infections alone are quite distinctive. It must be remembered, however, that there may be an association of these diseases with atrophic rhinitis. It is easy to differentiate the dry noses caused by surgical operations or cauterization.

In the cases of so-called one-sided atrophic rhinitis caused by extreme deflections of the septum, there will be found an attempted compensatory enlargement of the lower turbinate and that the discharge and crusts are caused by sinusitis. We are not dealing with a true atrophic rhinitis.

PROGNOSIS.

All the literature written in the past has given no encouragement in the prognosis. I wish to bring before you the fact, as stated in our book, "Intranasal Surgery," published in 1925, that the prognosis is good, if the case is properly treated.

TREATMENT.*

The sheet anchor of the treatment is the use of dessicated suprarenal gland (Armour). Two grain tablets are prescribed, to be taken three times a day, preferably at meal time. Only the dessicated gland should be used. Other gland therapy may be combined, if necessary.

If there is an ethmoiditis, the ethmoid capsule should be exenterated, but the middle turbinate must not be disturbed. All

*The treatment suggested is the one followed by us for the past fifteen years at the University Dispensary, the Minneapolis General Hospital, and in our private practice.

ostia of the sinuses should be patulous, and if not, should be made so. Intranasal window resections should be performed upon the antra if necessary, but the lower turbinate must be preserved. An external operation on the sinuses is not indicated.

If hypothyroidism is present, the dessicated thyroid gland, one grain tablet, is given, to be taken three times a day. The patient must be watched carefully to prevent the accumulative effects of the gland. Anemia or any other dyscrasia should receive attention. Hydrargyrum protoiodid tablets, grain one-tenth, given three times a day, stimulate the action of the endocrines.

A nasal oil should be dropped in the nostrils twice a day, the patient being in a supine position.

Packs of ichtholdine (Mulford) or a 3 per cent tincture of iodin in glycerin is indicated, to soften the crusts and stimulate the mucous membrane. Nasal douches of saline solutions are unnecessary.

Improvement is noticed within a very few days, and any odor, if present, will disappear in from one to two weeks, providing the sinuses are not invaded by the ozena germ. The patient will first notice the ease of cleansing the nose of crusts, the improvement in the general well being and in the mental attitude. The mucous membrane will become pink and the secretion will improve towards its normal character, due to the stimulation of the circulation and of the cells.

The gland treatment should be continued for some months or until there is a normal secretion of the endocrines, indicated by the disappearance of all symptoms.

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DISCUSSION.

Dr. Hal Foster, Kansas City: I consider it a great honor and pleasure to open the discussion of Dr. Pratt's paper on this important subject. Dr. Pratt has been giving much study to this condition for the last fifteen years. I have been much interested in the condition as it occurs in different races. When I first began to study and practice rhinology Professor Bostworth of New York and Professor Killian in Berlin talked about atrophic rhinitis. I had three or four cases a year. For the last five years I have not seen so many cases of this kind, since physicians have been removing pharyngeal adenoids.

Dr. Pratt states that perhaps chronic suppuration and infection of the posterior ethmoids might be a factor in the etiology of these cases. It seems to me that is very logical. I have tried to give vaccine treatment, and have tried to make the nose smaller by operation, but have stopped all that now, and I find it is better to clear up the posterior ethmoids and antrum. If that is done the malodor rapidly disappears and these young women are very grateful for that.

Dr. Pratt says he has never seen a case in a colored person. I have had opportunities, being born and raised among them, of examining many negroes, but I have never seen a case of atrophic rhinitis in pure negroes. A few years ago I spent a year among the Zulus in South Africa. I stopped at Port Moresby, the capital of New Guinea. They are all colored

people and I examined a number of noses down there. They had everything else you could think of—they are the most backward people in the world perhaps. Through the kindness of a British physician and the kindness of a physician in the hospital, I examined a number of these negroes and I did not find a case of atrophic rhinitis. I went to South Africa. I heard of one of the members of the Academy down there in New Guinea, a former President, who was down there taking pictures of the eyes of birds—Dr. Casey Wood. And I met another of our honorary members, Sir Lindsay Johnson, who has since died. The Zulus are fine specimens of humanity, and they had most everything else, but they do not have atrophic rhinitis. Plenty of nasal polypi, but no atrophic rhinitis. As far as my experience goes, I have never seen a case in the colored race.

I think the most important thing in these cases is to build these patients up. They are anemic; they need food with vitamines in it; they need spinach, oranges, liver. In the last six months an iron preparation has been put out that is very good in this condition.

Last summer I was in Moscow, and while there they tried to find some cases of atrophic rhinitis in the hospital for my benefit. We did find some cases among girls, but not a case in a man. I have seen only a few cases in men.

If Dr. Pratt's line of treatment is followed it will be of great benefit.

Dr. Harry L. Pollock, Chicago: It is always nice to hear new theories on a subject about which the etiology and pathology are not clear, and this is just another contribution to the hundreds of others on the etiology of chronic rhinitis. Dr. Pratt referred to my recent paper and I thought at that time that I really had advanced an important theory as to the etiology. In the ten cases I reported of atrophic rhinitis or ozena that were operated by Vogel of Berlin, the operation being for closure of the large space in the nose, the sphenopalatine ganglion was also removed and in every case there was complete degeneration of the sphenopalatine ganglion. I drew the conclusion therefrom which seems perfectly logical, that because the sphenopalatine ganglion supplies the mucous membrane of the nose and pharynx, atrophy of the ganglion would result in atrophy of the mucosa, or the picture presented by Dr. Pratt. The atrophy of the ganglion being present, one must speculate as to the cause of the degeneration. I attributed it to a sinus condition because every case has an ethmoiditis, as Dr. Pratt says. The other sinuses are involved in some cases, but in every one chronic suppurative ethmoiditis exists. In addition to atrophy there was also an inflammatory condition found in the ganglia.

Regarding the treatment, I would say that for the last twenty years we have had a large number of these cases on suprarenal extract and thyroid extract, and at the New Orleans meeting we gave a report on that treatment and the conclusion was that it had absolutely no effect. My theory is that atrophy occurs until there is complete atrophy of all the tissues due to degeneration of the ganglion, and in my experience the only treatment that does any good is to rid the nose of the large spaces. The damage has been done, the atrophy exists, and the odor is caused, as explained by Dr. Pratt, by the large inflow of air and contamination by

some saprophytic organism. To get rid of this infection, clean out the ethmoid sinuses, and as Dr. Pratt stated, there is always improvement, but not a cure. But if you fill these noses with ivory or some other transplant and make the size of the nose nearly normal, you will get a physiologic cure. I have had dozens of them in which all the symptoms were improved and many absolutely cured of all symptoms.

Dr. John A. Pratt, closing: I want to thank Dr. Foster for his discussion; also Dr. Pollock for bringing up the suprarenal extract. In the first place, suprarenal extract was not mentioned in the paper and I have never used it. I used the desiccated suprarenal gland only and I place particular stress that only the desiccated gland should be used. We have found no result whatsoever from the use of suprarenal gland extract. If you use thyroid, use the desiccated gland. We have found that Armour's suprarenal desiccated gland is the dependable one. If any one present will try out this method I am sure he will be satisfied with the results. I have never found a case that has not improved, and if response is slow, it is due to infection of the sinuses, and these should receive special treatment.

GENERAL AND OTOLOGIC ASPECTS OF CHOLESTERIN METABOLISM.

MAXWELL FINEBERG, M. D.,

ST. LOUIS.

Much research has been done in the attempt to prove a common relationship between arteriosclerosis, essential hypertension, chronic nephritis due to contracted kidney, apoplexy and chronic progressive internal ear deafness. The prevalent opinion until 1921 was that a primary arteriosclerosis formed the basic etiological factor. This interdependence of diseases formed a vicious circle which was not broken until 1921, when Volhard and von Bergmann[1] advanced their theory that essential hypertension should be considered a disease sui generis. Their theory has gained much universal credence in the last nine years, and it must be agreed that there is sufficient basis for their theory to better explain those clinical pictures which up to that time had to be explained by a vicious circle.

The work of Volhard and von Bergmann stimulated research to ascertain what might be the underlying cause of this new but very common disease. Verse,[2] in 1925, reported that he believed metabolic changes were primarily responsible for essential hypertension and that the metabolic changes might be the connecting link in the relationship between essential hypertension and those diseases which, prior to Volhard and von Bergmann's theory, had been attributed to primary arteriosclerosis.

Autopsy material was then examined more carefully for the presence or absence of arteriosclerosis in cases of this group of diseases. The reports were conflicting in that where the clinical history was suggestive of arteriosclerosis, the pathologists reported that there was little or no evidence of it. They frequently found the most marked evidence of arteriosclerosis in those cases which had no clinical mention of the condition.

Rosenblath[3] had previously shown that apoplexy should not be considered as due to gross hemorrhage in the brain but rather as due to numerous pin-point aneurysmal diapedesic bleedings. Westphal and Bär[4] elaborated on the work of Rosenblath and

came to the conclusion that arteriosclerosis has nothing to do with these diapedesic bleedings. They claim that both of these conditions, and hypertension as well, are the direct result of spastic vascular changes. But what is it that causes these spastic vascular changes? What is it that directly or indirectly causes arteriosclerosis, hypertension, apoplexy and spastic vascular changes? Is there some factor which may be the connecting etiologic link in these conditions?

The writer believes that there is a definite connecting link which may be found in the cholesterin metabolism in cases of arteriosclerosis, essential hypertension, apoplexy, chronic nephritis due to contracted kidney and progressive internal ear deafness. There is a very clearly marked hypercholesterinemia in all these conditions, and it is the opinion of the writer that the hypercholesterinemia is not at all coincidental, but that it plays no small part in the etiology of all these conditions.

There is quite definitely some close relationship between arteriosclerosis and chronic progressive ear deafness. The deafness was and is sometimes still considered due to an arteriosclerosis of internal auditory vessels. Alexander,[5] in 1902, actually demonstrated the sclerotic changes, and since then it has been taken for granted that arteriosclerosis produces an internal ear deafness. This, however, does not explain those cases which do not show any generalized arteriosclerotic changes; nor does it explain why so few specimens of sclerosis are reported in the literature.

There are numerous theories cited for the cause of chronic progressive internal ear deafness. Boenninghaus[6] believes that senile deafness and presbyacusis are caused by a degenerative process in the accoustic nerve. He says that one frequently finds a nerve impairment of hearing in premature arteriosclerosis which is clinically manifested by shortened bone conduction and a lowering of the upper tone limit. On the other hand, in cases of presbyacusis one frequently finds an irritative condition of the accoustic nerve, as manifested by head noises and dizziness. Thus Boenninghaus concludes that presbyacusis has to do with an accoustic neuritis which may often be caused by the same factors which cause an early arteriosclerosis.

Zwaardemaker[7] was among the earliest to investigate senile deafness. He concluded that the lowering of the upper tone limit was a normal physiological phenomenon which began in the third decade and then gradually increased.

Sporleder[8] thought that severe and even middle grade deafness must be produced by factors other than age. O. Mayer[9] suggests that "marasmus senilis" may be responsible for changes which are beyond physiological limits of normal. Lederer[10] agrees with O. Mayer in substance, but says it is impossible to estimate the age at onset of these physiological phenomena. Stein[11] lays much stress on arteriosclerosis as being the cause of senile deafness, and he claims that there is an accompanying local anemia which accounts for head noises.

From these few references it will be readily agreed that there is still some important link missing in the chain connecting arteriosclerosis, hypertension and senile progressive deafness. In delving further into this problem, it was thought that the examination of apoplectic patients might shed some light on the relationship. Working in conjunction with Privat-Docent Josef Berberich of Frankfurt-am-Main, the writer examined fifty-eight cases of apoplexy with a view to ascertaining whether there were any defect in hearing, and if there were, whether it was associated with the apoplexy. The vast majority of our cases had a very definite hypertension. Much stress is laid on this accompanying hypertension because it is believed that the internal ear changes found were related more or less closely to this phenomenon. We[12] found rather uniformly in fifty-eight cases of clearly proven apoplexy that there was an internal ear deafness of varying intensity. Subjectively, the patients complained more frequently of head noises than of impairment of hearing. Because of the constant presence of hypertension in our cases and the frequency with which we met the symptom of head noises, we were led to make the following statement: a patient who came to the doctor complaining of head noises and who showed a hypertension should be considered a possible eventual apoplectic.

The deafness in these cases was bilaterally equal and had no relation to the age of the patient or to the apoplectic insult. What might cause such an internal ear deafness? The conditions which come into consideration are occupational deafness, intoxication, chronic poisoning, lues, diabetes, nephritis and atypical forms of otosclerosis. Our cases were institutional and therefore, by careful examination, we were able to rule out all of the mentioned possibilities. Nevertheless, we remained with an internal ear deafness which could not be satisfactorily explained. We therefore deduced

that the deafness must be associated in some way with the accompanying hypertension.

In discussing the work of Rosenblath, Westphal and Bär, Verse and others on the question of hypertension, the opinion was expressed that hypertension and hypercholesterinemia were closely related. The theory is now advanced that hypercholesterinemia may be the missing etiological link in the chain formed by essential hypertension, arteriosclerosis, apoplexy and chronic progressive senile deafness.

There is no doubt that these conditions are separate entities and yet have something in common. If it can be definitely proven that an increased cholesterin metabolism plays some rôle in the etiology of these various conditions, then new avenues of approach will have to be opened to study their cause and treatment. The purpose of this paper is to show that an increased cholesterin content of the blood produces changes which are common in the various conditions under discussion. For this reason a search was made of the literature for histological information in chronic progressive internal ear deafness. Politzer and Morpurgo[13] reported atheromatous changes in the arteria auditiva interna in a case of arteriosclerosis. At a later date Politzer,[14] in his textbook, states that atrophy of the accoustic nerve may arise following circulatory changes. Alexander[5] described the histological changes he found in a case of arteriosclerosis. Manassa[15] described the histological picture of chronic progressive internal ear disease and pointed out various locations where the atrophy and degeneration were most marked. He blamed arteriosclerosis for causing these changes.

Kashiwabara,[16] in a case of apoplexy, saw scattered hemorrhages in the internal ear. He could not demonstrate any breaks in the blood vessels, so he termed the hemorrhages diapedesic bleedings. In later years Rosenblath and Westphal and Bär proved that apoplectic hemorrhages into the brain were also of a diapedesic character. This finding of Kashiwabara may prove of great interest in the study of "Ménière's symptom complex."

Stein,[17] Jaehne,[18] O. Mayer,[9] Witmaack,[19] Steurer[20] and others report numerous cases which show similar changes. They advance either arteriosclerosis or hypertension as the basic cause.

Thus far, it has been possible to link up the various conditions under discussion. Chronic progressive internal ear deafness seems to be an outstanding feature of them all. The next problem is

to connect these conditions to what is advanced in this paper as a possible etiological factor, namely, a hypercholesterinemia.

At this point a brief discussion of cholesterin metabolism is in order. The cholesterin content of normal blood serum is fairly constant, varying between 12 and 18 milligrams per 100 cubic centimeters of blood. An hypercholesterinemia may be traceable to the following factors:

1. To nutritional increase from ingested food. The increase, however, is not constant and is of a transient nature.

2. To cell destruction. Due to this, the cholesterin depots become overloaded and a circulatory surplus ensues. This surplus may then form a synthesis with enterogenous resorbed bile. In either event, a higher serum cholesterin result. This theory was advanced by Hueck.[21]

3. To endocrine disturbances, especially those of the adrenal gland.

4. To abnormal changes in the sex glands. Castration increases the cholesterin content of blood serum. It is also raised in the latter half of pregnancy. Eufinger and Bader[22] were able to show that this increase remained some time in postpartem women who were of the nervous type.

5. To changes in the thyroid gland. Cretins show an increased blood serum cholesterin. This is in keeping with the experimental evidence of Leupold,[23] who fed thyroid to animals.

6. To constitutional factors. To this group belong the people whom Westphal terms "typus apoplecticus" because they have a hypertension, an increased blood serum cholesterin and a lack of resistance to inflammatory processes.

7. To hitherto unexplained factors of unknown origin.

Westphal[4] propounded an interesting theory to explain how cholesterin maintains smooth muscle tone. He experimentally showed how a strip of arteriole contracts more vigorously in a solution containing cholesterin. Westphal then examined patients with hypertension and found that 89 per cent of them had a hypercholesterinemia. From his investigations he formulated the following conclusions:

1. Hypercholesterinemia and hypertension are closely associated.

2. An increased cholesterin content is found in young patients suffering from increased blood pressure, so that—

3. Arteriosclerotic contracted kidney cannot be a cause, but more probably is a sequel of the disease.

From these deductions an explanation may be found for numerous transient conditions—e. g., Ménière's, vague attacks of dizziness, etc.

It is not to be wondered at that definite histological changes in temporal bones of beginning senile deafness have hitherto not been demonstrated. The early symptoms depend on functional changes in the labyrinthian vessels. Only when the disease has been of long standing is it possible to histologically show a continued contractile condition of the vessels together with a cholesterin deposit. At this stage the changes are similar to those found in the kidney, spleen and pancreas.

In the work previously referred to,[12] we reported on the histological findings in nineteen cases, in six of which we had been able to do hearing tests shortly before death. All six cases showed an internal ear involvement of varying degree. The nineteen cases were comprised of seven apoplectics, six diabetics, two arteriosclerotics, one pyemic and two uremics following chronic nephritis and one case of frontal sinus empyema. The ages of these patients varied between 49 and 85 years.

The temporal bones were prepared in accordance with Witmaack's method and then embedded in gelatin. Serial sections were cut and then stained with scarlet red and hematoxylin.

At this point it should be explained that various terms have been used in the designation of identical histopathological changes. Thus, in this article, the terms lipoid deposit, fatty lipoid deposition, fatty deposit, fatty or lipoid changes are all used to designate different degrees of the same pathological entity.

The histological changes found in all nineteen cases were similar, so we shall only point out individual factors of interest in the group. A typical arcus lipoides myringis (Fig. 1) is seen in nearly every case. The lipoid deposits in the tympanic cavity are seen only in the area of the tube opening. One never sees lipoid deposits in the submucous layers except where there are co-existing inflammatory changes. The inflamed mucous membrane has undergone some fatty change. (Fig. 2.) The lipoid deposit is seen only in the newly formed inflammatory tissue and cannot be seen in the intact normal tissue. The ossicles and secondary tympanic membrane do not show any characteristic lipoid changes.

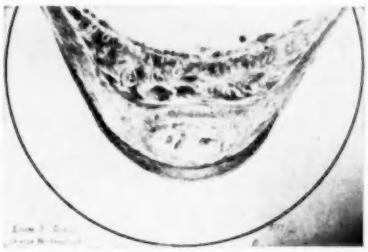

Fig. 1. Arcus lipoides myringes and hammer handle in 66-year-old diabetic.

Fig. 2. Otitis media in 28-year-old woman with nephritis and hypertension. The inflamed mucous membrane has undergone fatty changes.

The lining of the mastoid cells normally shows no fat except for normal fatty medullary tissue. However, lipoid changes occur in our cases where an otitis was co-existent. (Fig. 3.) One of our cases died of meningitis following labyrinthotomy, and the newly formed granulation tissue showed the typical lipoid deposits.

Our cases with co-existent inflammatory changes show, without question of doubt, that when the blood contains an increased amount of cholesterin, it will be deposited in newly formed connective tissue and cellular elements. This confirms the belief that the process has to do with an equalization or stabilization of the vital storehouses of the reticulo-endothelial apparatuses.

Fig. 3. Lipoid changes in a case of mastoiditis. Cellular exudate shown in small upper section.

Fig. 4. Bony cells of facial canal show ring-form storehouse of fat and lipoids.

The chorda tympani and facial nerve were unchanged. The bony cells of the osseous facial canal showed a definite ring-form storehouse of fat and lipoids. (Fig. 4.)

The cochlea showed in every case, but more particularly in the diabetics, some lipoid deposits in the stria vascularis and its epithelium. The capillaries are partially filled with fat and the pigment of the epithelium is no longer to be recognized. (Fig. 5.)

The prominent vessel between the stria vascularis and the external spiral sulcus shows in particularly nice fashion what happens in a hypercholesterinemia. The wall is covered by fat, exactly as we find it in the kidney, spleen and other organs.

The cells of Claudius show changes only in the advanced diabetics. The cells of Hense and Deiter show lipoid deposits which are more marked in the cells nearest the lumen of the ductus cochlearis.

The membrana tectoria shows no changes. The internal spiral sulcus, the membrana basilaris, the ligamentum spirale, the stria vascularis and Reissner's membrane show characteristic lipoid deposits.

The vestibule and semicircular canals show no histological changes.

In summing up our histological findings, we may say that our nineteen cases of apoplexy, diabetes, hypertension and nephritis, all showed a typical lipoid infiltration in the drum membrane, in the ossicular chain joints and in the inflamed mucous membrane of the middle ear and mastoid process. All of our cases show a typical lipoid infiltration in the isolated elements of the ductus cochlearis, of the ligamentum spirale and of the stria vascularis. The small arteries and precapillaries show changes which are similar to arteriosclerosis. Thus we conclude that an increased cholesteria content in the blood produces a lipoid change in the peripheral auditory end organ.

It was stated earlier that the object of this paper was to link up hypercholesterinemia with arteriosclerosis, apoplexy, hypertension, chronic progressive internal ear deafness and chronic nephritis. So far it has been clearly shown by historical, clinical and pathological proof that a hypercholesterinemia forms a basic link in the chain of diseases we have discussed. The only additional proof available would be experimental. For this reason it was attempted to produce a hypercholesterinemia in rabbits in

order to determine if an artificial hypercholesterinemia could produce the same histopathological changes that we described in our nineteen histologically examined cases.

We[12] fed chemically pure cholesterin to twenty-six rabbits whose average age at time of starting was three months. Feedings were given by the stomach tube. Ten animals at random were castrated, in order to determine how much the hypercholesterinemia is increased by this measure. The cholesterin was put in oil, 20 grams of cholesterin to 100 cc. of oil. The animals were fed three times a week, starting with 5 cc. and rapidly increasing it to 10 cc. per feeding. We fed sixteen of our animals for more than 100 days. The average feeding time was seventy days. The longest time was 227 days, which animals received

Fig. 5. Reissner's membrane shows fine fatty droplet formation in the elastic fibers. The stria vascularis and capillaries show lipoid deposits.

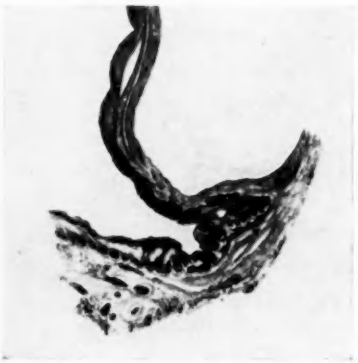

Fig. 6. Experimental arcus lipoides in cholesterin fed rabbit.

184 grams of cholesterin. Many animals died suddenly during the feedings. We killed sixteen animals immediately after feeding and allowed five of the 227-day animals to live for three months after the last feeding. The reason for this exception was to investigate to what extent a long produced hypercholesterinemia is transient. Macroscopically, the arcus lipoides corneæ gradually disappeared, and microscopically the fatty deposits seem actually to have melted away.

The normal blood serum cholesterin of rabbits swings between .05 gm. and .09 gm. per 100 cubic centimeters. The repeated examinations of our rabbits revealed that in as short a time as fourteen days the average cholesterin content was 1.2 gm. per 100

cc., and at 60 days it was 2.0 gms. The castrated rabbits showed a definite proportional increase.

We examined the blood pressure in five of our animals at random and were able to confirm Westphal's[4] finding that artificial hypercholesterinemia is accompanied by hypertension.

The temporal bones were fixed and examined with the identical technic used in the human specimens. The control animals showed no fatty or lipoid changes in the ear.

Our group of sixteen rabbits all showed practically the same histopathological changes with a difference of degree proportionate to the period of time of cholesterol feeding.

Fig. 7. Experimental lipoid deposit about annular ligament of stapes.

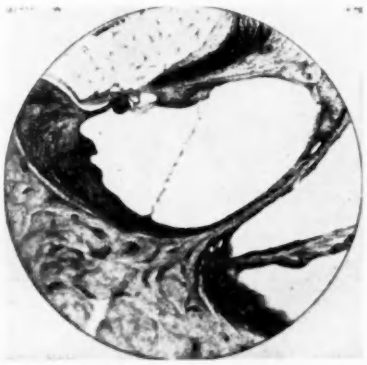

Fig. 8. Cochlea in cholesterin fed rabbit. Lipoid deposit is marked in the stria vascularis, ligamentum spirale and in the cells of Hense, Claudius and Deiter.

Cerumen in the external auditory canal showed a high degree of lipoid deposit. This may or may not be within normal limits. The cartilage of the external ear, however, showed a tremendously high degree of lipoid in droplet formation. The nuclei of the cells are almost obliterated.

The drum membranes show a quite characteristic fatty deposition with a diffuse lipoid deposit in all parts of the drum. (Fig. 6.) The mucous membrane of the middle ear shows scattered lipoid deposits, but the capillaries of the mucosa contain a serum heavily laden with lipoids.

The bone marrow of the ossicles and the ligamentous joints show, just as they do in the human, a slight deposit in the periph-

eral parts and a more markedly diffuse one about the annular ligament of the stapes. (Fig. 7.)

The secondary tympanic membrane shows no definite changes. The remainder of the bony parts show a marked lipoid increase in the marrow, and the capillaries are filled with serum containing lipoids.

Muscles and connective tissue show a fine fatty droplet deposition.

There are no changes to be seen in the facial nerve itself, but the bony cells of the facial canal show an annular deposit similar to that seen in the human sections. There are no deposits in or

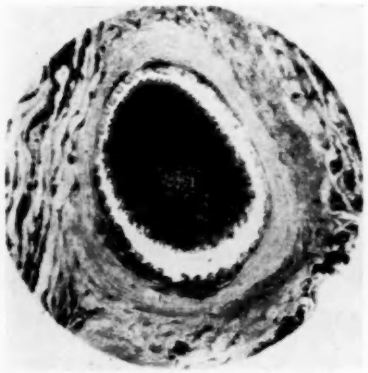

Fig. 9. Intima of internal carotid artery shows lipoid deposit in cholesterin fed rabbit. This is quite similar to an arteriosclerosis of the vessel.

Fig. 10. Modiolus of cholesterin fed rabbit showing vessels filled with lipoid.

about the membranous and bony semicircular canals of the rabbits, which differs from the ring-like deposits noted in the human.

The cochlea shows marked fatty deposits in almost all parts. (Fig. 8.) The deposit is most marked in the stria vascularis, the ligamentum spirale, the sulcus spiralis externus and the cells of Claudius, Hense and Deiter. Reissner's membrane, the ductus cochlearis, the ganglion spirale and Rosenthal's canal show only sparse infiltration. The intima of the internal carotid artery shows a marked fatty infiltration. (Fig. 9.)

Fig. 10 shows vessels in modiolus of cholesterin fed rabbit. They are filled with lipoid.

The animals that were killed three months after the last feeding show little or no histological evidence of lipoid deposit.

The experimental evidence obtained from an artificial hypercholesterinemia establishes clearly that cholesterin metabolism plays a large rôle in certain types of internal ear deafness.

In conclusion, it is the writer's belief that the systematic clinical, anatomical, pathological and experimental investigations presented in this paper establish the importance of cholesterin metabolism and supplies a probable clue to the missing link which connects arteriosclerosis, essential hypertension, apoplexy and internal ear deafness.

AUTHOR'S NOTE.—The original illustrations for this article were made in color (lumière) plates which show differentiation very much more clearly than the black and white reproductions seen in this article. Indulgence is asked if the reader is unable to make out the details as outlined in the text.

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DISCUSSION.

Dr. S. J. Kopetzky, New York City: Dr. Fineberg states that in the autopsy material that was available to him in cases in which he had previously established an internal ear involvement associated with hypertension and arteriosclerosis, characteristic findings were present histologically in the form of lipoid and fat deposits in the middle and internal ear. These findings led him to believe that they in some way interfered with the function of the inner ear and he was able to reproduce experimentally the identical pathological changes by inducing a hypercholesterinemia in rabbits. He also states that in these rabbits wherein a normal diet was re-established, no such lipoidal deposits could be demonstrated. In other words, these experimentally produced deposits could be demonstrated only while the hypercholesterinemia was present; with the disappearance of the hypercholesterinemia the tissues of the internal ear returned to normal.

It is of the utmost importance in a work of this kind where conclusions are drawn of the close relationship of an increased cholesterin content of the blood to senile deafness, and internal ear deafness, in cases of hypertension and kindred conditions, to state whether or not the changes observed histologically are degenerative changes of the tissue, or whether the lesion observed is merely a transient deposit of a foreign substance The experimental work of Dr. Fineberg points toward the belief that, in his rabbits, the pathological lesion of lipoidal deposits is temporary, and that with the reestablishment of the normal cholesterin content in the blood the tissues in the internal ear returned to normal. In other words, function would be only temporarily impaired, and we can deduce therefrom

that just as in other tissues the body removes foreign material from the inner ear as well.

If such a state of affairs were present in the human being we must believe that a disease which will produce a hypercholesterinemia will produce deafness which will disappear as soon as the metabolic disturbance is rectified. On the other hand, Dr. Fineberg's autopsy material on the human, in cases of senile deafness and internal ear deafness associated with hypercholesterinemia, would tend to show that the lesion is a permanent one. This makes me believe that the difference between his experimental work and his work on the human lies in the following factor: the changes noted in the human were due to fatty degeneration of tissue, whereas the changes in his rabbits were due to the deposits of lipoidal substances in otherwise healthy tissue.

I realize that the point I am bringing up is purely academic, but if we are to accept the relationship of cholesterin in metabolism to the development of internal ear deafness, we must have proof other than the experimental portion of Dr. Fineberg's work. His work on the human material is far more convincing than his experimental work; but even in this the diseases which he has studied are degenerative diseases and the findings may be the result of local trophic disturbances rather than of metabolic origin.

Dr. Maxwell Fineberg, closing: Dr. Kopetzky is probably right. The paper was not intended to provoke any argument on Dr. Kopetzky's point, which is well taken. However, we are at a standstill until we can get more detailed information as to the degenerative and atrophic changes in the human ear, to better explain the temporary changes in the animal. At the present time we are attempting to find a clinical substance that will render cholesterin inert. We would like to be able to rule out the temporary phase in the rabbits by giving them some such chemical. If the findings then disappear, I think it will prove Dr. Kopetzky's point.

EARLY INTRINSIC CANCER OF THE LARYNX, DIAGNOSIS AND TREATMENT: OBSERVATIONS ON LARYNGOFISSURE AS A METHOD OF TREATMENT IN A SERIES OF CASES.

GABRIEL TUCKER, M. D.,

PHILADELPHIA.

"The accurate study of laryngeal cancer has been possible only since 1855, when Manuel Garcia invented the laryngoscope."

Today we have the improved and perfected laryngoscope after the model of Garcia, and by its use laryngologists should be able to detect a lesion in the larynx and, as Dr. Delavan has so well said, "by subconscious reasoning based upon long experience of many closely observed cases," make a correct diagnosis of *early* cancer of the larynx. Every otolaryngologist who is especially interested in the larynx will verify, however, the fact that today the diagnosis is rarely made early by the rank and file of physicians who first see these patients. The principal reason for this is that the great majority of physicians do not take the time to acquire the technic required for accurate visualization of the larynx. In the rush of routine work a hoarse patient, after an imperfect visualization of the larynx, is given a gargle, or, worse still, a local application of silver nitrate which will produce a burn and mask the appearance of the disease in the larynx that might lead to proper diagnosis. The patient is told that he has a chronic laryngitis and is treated for this until the cancer is far advanced.

As a means of accurate diagnosis in a laryngeal lesion that has been studied properly by mirror examination and the routine diagnostic studies which are so well known that they need not be repeated, we have a method of examination which should be used supplementary to the mirror as the final step in differential diagnosis, namely, direct laryngoscopy and biopsy by the method of Chevalier Jackson. By this method every part of the larynx can be seen and tissue removed accurately from any location. (Fig. 1.)

The length of this paper will not permit a full discussion of intrinsic cancer of the larynx, but every physician, and more par-

Fig. 1. The Jackson anterior commissure laryngoscope, and basket tissue forceps, in correct position for removal of specimens of tissue from the larynx. With the patient in dorsal recumbency under local anesthesia, tissue may be removed from any portion of the larynx desired.

ticularly every laryngologist, should read carefully "Cancer of the Larynx," by Sir St. Clair Thomson, the dean of British laryngology, and his very able associate, Mr. Lionel Colledge. In large part, due to the teaching and practice of Sir St. Clair Thomson in the British Isles and of Dr. Chevalier Jackson in the United States, American laryngologists are realizing that intrinsic cancer of the larynx, if recognized early, is, in the majority of cases, in such a location that it can be removed surgically without the removal of the larynx, with a very high percentage of cure. Early diagnosis, then, is "the pearl of great price" in the cure of intrinsic cancer of the larynx. In the cases that are not amenable to laryngofissure early diagnosis is also of very great importance in order that laryngectomy may be done before metastasis takes place. We may speak, then, of intrinsic cancer of the larynx as being early if a diagnosis can be made before metastasis has occurred. Intrinsic cancer of the larynx occurs most often in the "anterior-intrinsic area" (Jackson). This area includes the anterior two-thirds of the true vocal cords, the adjacent portions of the ventricles of the larynx, and the anterior commissure. (Fig. 2.)

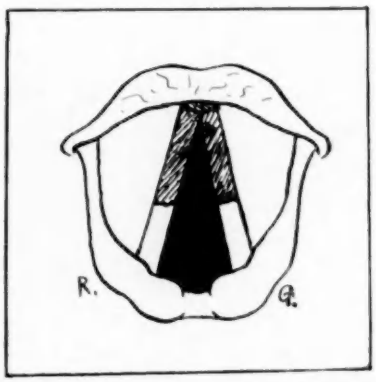

Fig. 2. Schematic drawing of the larynx. The shaded area represents the "anterior intrinsic area." Cancer limited to this area, on either side or the anterior commissure is amenable to cure by laryngofissure.

A growth occurring in this area is amenable to removal by laryngofissure because of the tendency to late metastasis due to the peculiar lymphatic arrangement. (Cuneo.) (Fig. 3.) Also, this is the area in which the lesion is most apt to be overlooked in mirror examination because of the difficulty in many patients in getting a clear view with the mirror of the anterior intrinsic area.

Diagnosis: Chronic hoarseness is a constant symptom in practically every case of intrinsic cancer of the larynx. A growth involving the ventricular band, according to Sir St. Clair Thomson, may produce only slight impairment of the voice. There is, however, much more frequently a sticking sensation, or actual pain, which should lead to careful examination and a correct diagnosis of early involvement. This applies also to lesions in the deep subglottic area. The treatment of involvement in the subglottic area and ventricular bands is not so successful by laryngofissure unless the lesion is of very limited extent.

The routine diagnostic procedure which has been so often repeated in teaching and so infrequently carried out in practice in the individual case, should be, first, careful examination and routine study of the larynx with a mirror of variations from normal in color of mucous membrane, in motility, and in the contour of the larynx should be made carefully. Second, the general examination of the patient should include a careful history, a general physical examination, careful roentgen-ray study of the neck and

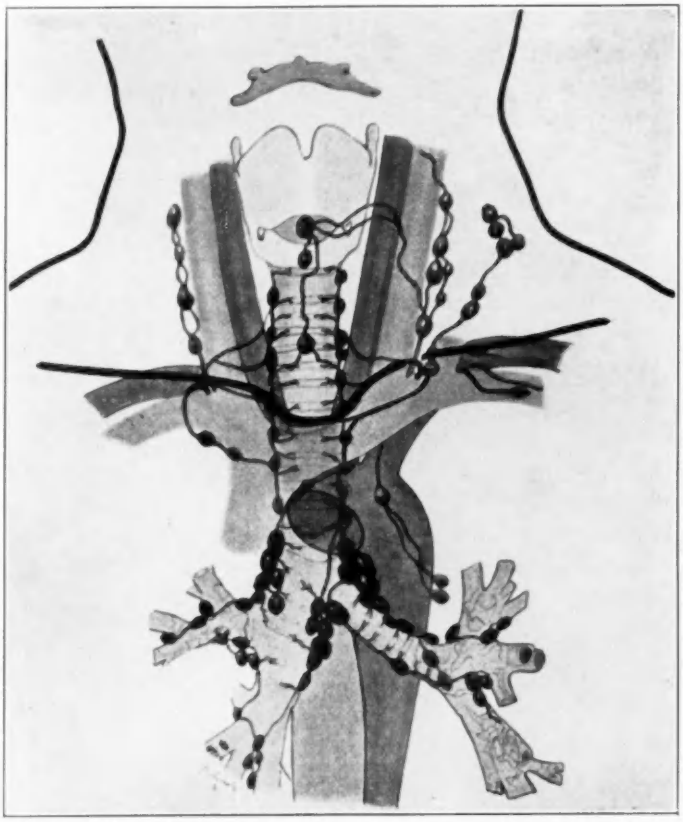

Fig. 3. Lymphatic glands of the larynx, trachea and bronchi. (Professor Dr. M. Hajek, after Thompson and College, London.)

chest, blood and serological studies, in order that we may determine the relation of general or systemic disease to the local lesion in the larynx. The final step in differential diagnosis should be direct laryngoscopy and biopsy.

Biopsy: There have been many objections to biopsy, the first one being the fallibility of the pathologist's report. The report of the pathologist should always be taken in conjunction with the clinical findings in the case. If it is a question of the interpretation of the histologic picture, any pathologist will welcome a consultation with a peer. The complaint has been made that the pathologist pronounces the condition cancer when

it is not. That has not been my experience. Another objection to biopsy has been the possible production of metastasis by biopsy. This is very well refuted, however, by the fact that we can get a report from a fixed specimen, in a period of 48 hours, certainly before there could be opportunity for metastasis to occur. All our pathologists agree that there have been no bad results from this procedure when properly performed. Biopsy by direct laryngoscopy gives a certainty of location and accuracy of removal that can be obtained in no other way. I have never regretted doing a biopsy for a differential diagnosis, but I have regretted very much my inability to get permission to do a biopsy until the lesion was far advanced.

Early biopsy will frequently reveal a precancerous condition. Operative removal in the stage when the lesion is precancerous will require a much less radical procedure to effect a cure than after the lesion is so far advanced that its nature is perfectly evident on mirror examination. In a recent case the author found both tuberculosis and cancer in the same microscopic section. (Figs. 4, 5 and 6.)

Pathology: McKenty gives the relative frequency of the different forms of carcinoma of the larynx as squamous carcinoma, 96 per cent; basal cell carcinoma, 2 per cent; papillary carcinoma, 1 per cent; adenocarcinoma, 1 per cent. The careful grading of the cancer, according to Broder's classification, is advocated by Gordon New as a means of determining the degree of malignancy. Not all pathologists, however, follow this method of grading, and clinically we must depend more upon the location and the extent of the lesion as found on mirror and direct examination than on the grading of the histologic characters. It would seem advisable to remove a growth at laryngofissure, even if of the highest grade of malignancy, if it was in a favorable location and of limited extent.

It is generally recognized that cancer of the larynx is not highly malignant in type, and if the cases are closely followed after laryngofissure, a laryngectomy can be done if recurrence takes place. This gives the patient a chance to keep his larynx and no harm results. (See case 25 in series.)

A series of 30 cases of laryngofissure for early intrinsic cancer of the larynx from the bronchoscopic clinics of the University of Pennsylvania, operated upon by the author, is presented in

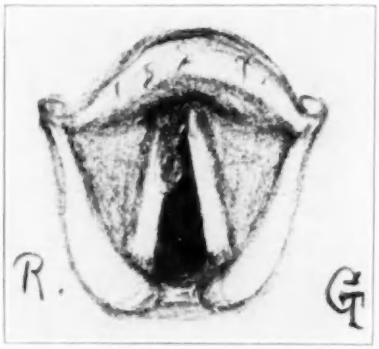

Fig. 4. Sketch of mirror image of the larynx showing irregular ulcerating mass of tissue occupying the anterior third of the right cord. Direct examination showed the lesion to extend across the anterior commissure to the under surface of the left cord. The other portions of the larynx were free from involvement.

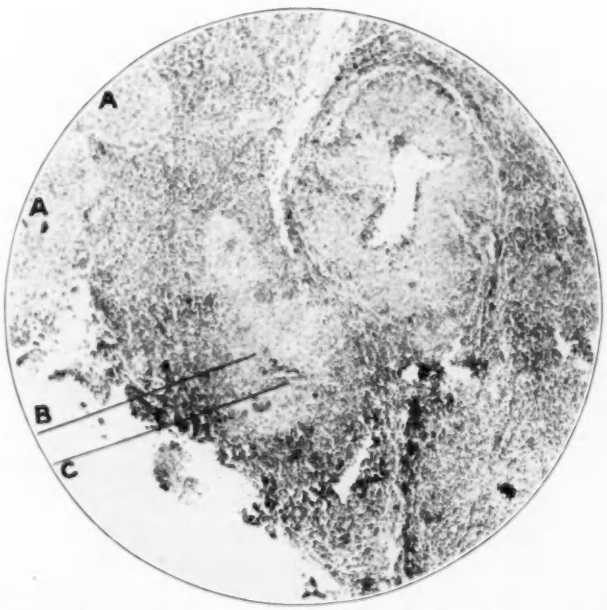

Fig. 5. Low power microscopic section from biopsy specimen showing areas of cancer and tuberculosis: *A, A,* cancer; *B,* tuberculosis; *C,* giant cell.

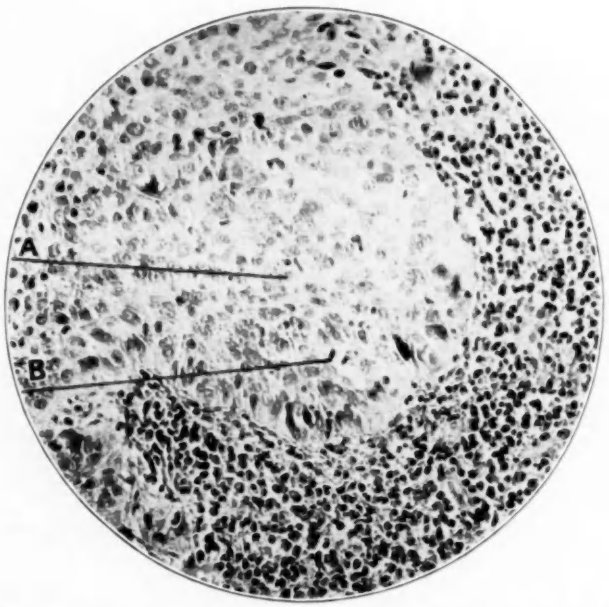

Fig. 6. High power view of cancer area (*A*) showing mitotic figure (*B*). Dr. E. A. Case.

tabulation. (See table.) There was no operative mortality in the series. In twenty-five cases over one year has elapsed since the operation. There was no operative mortality in the series. In twenty-five cases over one year has elapsed since the operation. There have been two recurrences in this group. One recurrence occurred nine months after laryngofissure; the patient refused laryngectomy and died at the end of one year after operation. The second case had a recurrence at one year and five months after partial laryngectomy, and a second recurrence ten months later, which will require laryngectomy. Five cases in the series are recent, under one year, have no recurrence, but are not considered in the percentage estimate. The longest duration of cure is seven years; eight cases are of over four years' duration.

The technic of laryngofissure used in the series follows very closely that of Chevalier Jackson. Thyrochondrotomy of Sir St. Clair Thomson was employed in several cases.

In the first half of the series general anesthesia, intratracheal ether, was used. At the present time local anesthesia only is employed. Technic of laryngofissure: The patient is carefully pre-

pared after a diagnosis of cancer has been made by biopsy. Blood chemistry, kidney function tests and general examination by a competent internist are carried out. Preliminary morphin and atropin is not administered unless the patient is very apprehensive or of a particularly nervous temperament. The patient is placed in dorsal recumbency with a sand pillow beneath the shoulders with the head slightly lowered. One-half per cent novocain is infiltrated into the front of the neck in the median line from the hyoid bone to the suprasternal notch.

The front of the larynx, cricoid and upper tracheal rings are exposed. Great care is used not to skeletonize the larynx or to injure the external perichondrium of the thyroid cartilage. Bleeding points are ligated. The larynx is then opened by incising the cricothyroid membrane. Through this incision is introduced a small bronchoscopic sponge, saturated with 10 per cent cocain. The interior of the larynx is swabbed with this solution, sufficient being applied to produce anesthesia. If the cancer is limited to one cord and does not involve the anterior commissure the blade of a turbinotome is inserted through the incision in the cricothyroid membrane and is carried upward inside the larynx until it is in contact with the base of the epiglottis. The outside blade being level with the thyroid notch. The thyroid cartilage is then clipped through, the wings retracted, the growth on the cord exposed and carefully examined. Then an incision extending through the mucous membrane completely surrounding the growth and about a half centimeter beyond the furthest extension of the growth is made. A careful dissection is then made, elevating the internal perichondrium of the thyroid wing on the affected side from the cartilage. A curved scissors is then used to cut through the mass of tissue from the incision in the mucous membrane, including the internal perichondrium, that has been dissected free. Hemorrhage is controlled by ligation and by suture ligature and pressure.

In the anterior commissure growth, the technic of Jackson (see Fig. 7) is followed. The cartilage is sawed through, using a Clerf saw. Great care is taken not to penetrate the internal perichondrium. To avoid this a subperichondrial dissection may be made by dissecting downward from the thyroid notch and upward from the lower border of the thyroid cartilage until the internal perichondrium is free from the anterior angle of the thyroid. A Gigli saw may be passed between the internal perichondrium

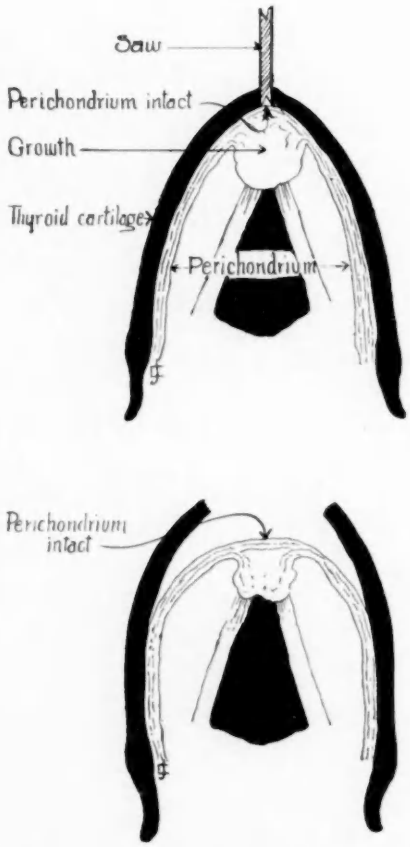

Fig. 7. Jackson's method of subperichondrial dissection in anterior commissure growths. Procedure in cases in which the cancerous growth is in the anterior commissure. The shears are not used for fear of cutting the growth. The thyroid cartilage is sawed through without injury to the inner perichondrium which is then dissected backward safely beyond the posterior limits of the growth.

and the cartilage, and the cartilage sawed through from inside, if desired. The thyroid wings are retracted (See Fig.) and the internal perichondrium separated to a point well beyond the margin of the growth, and the cancer bearing growth area with a wide margin of normal tissue is excised. Hemorrhage is controlled, and in cases where considerable of the mucous membrane of the epiglottis has been excised it is advisable to place a deep suture from the incision on either side just above the thyroid notch, passing beneath the base of the epiglottis. When this suture

is tied it holds the base of the epiglottis forward and prevents it dropping back toward the posterior commissure and helps to maintain a good lumen through the glottis. Hemorrhage inside the larynx must be very carefully controlled before the larynx is allowed to close. This is usually not a difficult matter to accomplish, but should be very carefully done. The external wound is closed by interrupted silkworm sutures which include the skin and subcutaneous tissue and muscle down to the cartilage, but not including the cartilage. A strip of iodoform gauze is placed at the lower angle of the wound, securing it with a suture so that it cannot get into the larynx through the opening in the cricothyroid membrane. A second strip of gauze is placed in the middle of the incision beneath the sutures over the edges of the cartialge. The upper drain is removed in twenty-four hours. The lower angle of the wound is kept open until the incision in the cricothyroid membrane closes. Abundant dressings are applied to the external wound and are changed frequently as required. The patient is placed in bed with a backrest. If there is difficulty in swallowing, a small rubber catheter is passed through the anterior nares into the upper esophagus. Abundant fluids are given. Elderly patients are gotten out of bed within the first twenty-four hours. The sutures are removed, usually within four or five days. If infection develops in the wound, all sutures are removed. On appearance of infection, the wound is opened and is allowed to heal by granulation. If this occurs the convalescence is usually lengthened from ten days to three weeks. Otherwise the end result seems to be the same.

Complications.—Postoperative hemorrhage is a possible complication, and provision is made by this technic for the insertion of the tracheotomy tube through an incision of the tracheal rings which have already been exposed. The larynx may then be packed above the tracheotomy tube. The patient is encouraged to cough, and it is of very great assistance to the patient for him to place his hand over the outside of the dressing and hold the wings of the thyroid cartilage together when he attempts to cough. Morphin and all cough sedatives are very carefully avoided. Special precaution must be taken in regard to this, for all resident physicians are trained to give morphin postoperatively, and it must not be given to patients who have had external surgery of the larynx.

In a considerable percentage of the cases granuloma will form in the anterior commissure after the wound has closed externally. Occasionally a small piece of cartilage will necrose and separate. This cartilage can be removed externally or by direct laryngoscopy. The granulomatous masses may require direct laryngoscopic removal several times during the first three or four months. It requires only a few minutes and is done with local anesthesia. The removed tissue is, of course, submitted to histologic study to be sure that we are not dealing with a recurrence. Tracheotomy is not done unless it is required to pack the larynx on account of hemorrhage or some undue reaction in the larynx.

The Technic of Sir St. Clair Thomson.—The essential difference in Thomson technic is that the thyroid wing on the affected side is dissected free of perichondrium, both internally and externally, and the freed portion of cartilage excised. This gives a much better exposure of the inside of the larynx and furnishes an excellent approach for partial laryngectomy if it is desired to carry out this procedure. In a number of cases not enumerated in this series a partial laryngectomy has been done by this method, and in some instances radium applications made inside the larynx, allowing the larynx to remain partially open, with excellent results.

CONCLUSIONS.

1. Early intrinsic cancer of the larynx will be recognized when the profession realizes fully the possible significance of chronic hoarseness. Biopsy by means of direct laryngoscopy should be the final step in the diagnostic study.

2. Early "anterior intrinsic cancer" of the larynx is amenable to cure by laryngofissure, which will save the patient's larynx as well as his life, with no operative mortality. The anterior intrinsic group includes a large percentage of all cases of cancer of the larynx.

3. Laryngectomy will cure practically all recurrences after laryngofissure if the cases are properly followed.

4. In twenty-five cases of the series reported, over one year has elapsed since laryngofissure. There have been two recurrences in two patients, giving a percentage of lasting cure of 92 per cent to September, 1931. One case of recurrence is well, without metastasis, following laryngectomy.

5. Partial laryngectomy with postoperative radiation has given good results where there was a contra-indication to laryngectomy in several cases not in this series.

6. Laryngectomy will cure a large percentage of posterior intrinsic cancer if the diagnosis is made while the lesion is still early.

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THYROTOMY OR LARYNGOFISSURE IN THE CURE OF EARLY INTRINSIC CANCER OF THE LARYNX.

Case Series From University of Pennsylvania Bronchoscopic Clinics.

TABULATION OF CASES.

Series No.	Age and Sex Occupation	Duration of Symptoms	Location of Lesion	Date of Biopsy	Thyrotomy date Anesthesia	Convalescence Complications	Recurrence Metastasis	Result	Period of cure, to Sept. 1931
1.	Male, 64 yrs. Plumber	Hoarseness 6 months	Anterior 3rd right cord	Squamous epithelioma Aug. 1, 1924	Aug. 5, 1924 Intra-tracheal ether	2 weeks granuloma	None	Voice clear Airway normal	7 years and 1 month
2.	Male, 33 yrs. Broker	Hoarseness 3 years	Entire right cord and ventricle	Inflammatory epithelioma May 7, 1925	May 29, 1925 Intra-tracheal ether	3 weeks granuloma	None	Voice hoarse Airway normal	6 years and 4 months
3.	Male, 60 yrs. Brick worker	Hoarseness 18 months	Anterior 3rd l. commissure	Squamous epithelioma Oct. 11, 1926	Oct. 15, 1926 Intra-tracheal ether	3 weeks	None	Voice hoarse Airway normal	4 years and 11 months
4.	Male, 54 yrs. Salesman	Hoarseness 1 year	Anterior 3rd left cord	Squamous epithelioma Oct. 11, 1926	Oct. 15, 1926 Loc. novocaine and cocaine	2 weeks	None	Voice clear Airway normal	4 years and 11 months
5.	Male, 56 yrs. Univ. Prof.	Hoarseness 4 months	Anterior commissure	Basal cell carcinoma Dec. 9, 1926	Dec. 11, 1926 Loc. novocaine	3 weeks granuloma	None	Hoarse voice Airway normal	4 years and 9 months
6.	Male, 70 yrs. Ins. Agent	Hoarseness 4 months	Anterior commissure	Squamous carcinoma March 8, 1927	March 15, 1927 Intra-tracheal ether	3 weeks	None	Hoarse voice Airway normal	4 years and 6 months
7.	Male, 65 yrs. Salesman	Hoarseness 8 months	Anterior commissure	Squamous epithelioma April 11, 1927	April 14, 1927 Intra-tracheal ether	2 months slight peri-chondritis	None	Voice good Airway free	4 years and 5 months
8.	Male, 44 yrs. Automobile Mechanic	Hoarseness 9 months	Mid-third left cord	Squamous carcinoma May 3, 1927	May 6, 1927 Intra-tracheal ether	4 weeks (3) granuloma	None	Good voice Airway normal	4 years and 4 months
9.	Male, 54 yrs. Civil Engineer	Hoarseness 8 months	Mid-third left cord	Papilloma beg. epithel.	Nov. 4, 1927 Intra-tracheal	4 weeks	None	Good voice Airway normal	3 years and 10 months

10.	Fem., 35 yrs. Housewife	Hoarseness 8 months	Anterior 3rd right cord	Papilloma beg. epithel. March 27, 1928	April 3, 1928 Intra-tracheal ether	4 weeks granuloma	None	Good voice Airway normal	3 years and 5 months
11.	Male, 68 yrs. Barber	Hoarseness 8 months	Mid-third left cord	Epithelioma April 24, 1928	April 28, 1928 Loc. Nov. & Coc.	4 weeks	None	Good voice Airway normal	3 years and 5 months
12.	Male, 57 yrs. Salesman	Hoarseness 2 years (intermittent)	Anterior 3rd right cord	Papilloma and early malig. April 20, 1928	May 3, 1928 Loc. Novocane	3 weeks granuloma	None	Good voice Airway normal	3 years and 4 months
13.	Male, 54 yrs. Salesman	Hoarseness 6 months	Mid-third right cord	Epithelioma May 10, 1928	May 12, 1928 Loc. Nov. & Coc	2 weeks granuloma	None	Voice clear Airway normal	3 years and 4 months
14.	Male, 57 yrs. R. R. Eng.	Hoarseness 7 months	Anterior two-thirds left cord	Epithelioma June 4, 1928	June 12, 1928 Loc. Novocane	2 weeks granuloma	None	Voice clear Airway normal	3 years and 3 months
15.	Fem., 57 yrs. Housewife	Hoarseness 1 year	Anterior two-thirds right cord	Epithelioma Sept. 10, 1928	Sept. 13, 1928 Intra-tracheal ether	4 weeks	None	Voice good Airway normal	3 years
16.	Male, 53 yrs. Carpenter	Hoarseness 5 months	Anterior commissure both cords	Inflammatory epith. change Oct. 30, 1928	Nov. 13, 1928 Loc. novocaine	4 weeks perichondritis	None	Voice good Airway free	2 years and 10 months
17.	Male, 63 yrs. Huckster	Hoarseness 5 months	Anterior 3rd left cord	Squamous cell carcinoma April 1, 1929	May 8, 1929 Loc. Nov. & Coc.	3 weeks granuloma	None	Voice good Airway clear	2 years and 4 months
18.	Male, 58 yrs. Minister	Hoarseness 8 months	Mid-third left cord	Squamous carcinoma May 15, 1929	May 18, 1929 Loc. Nov. & Coc.	3 months perichondritis & pul. T. B.	None	Voice good Airway free	2 years and 4 months
19.	Male, 64 yrs. R. R. Cond.	Hoarseness 11 months	Ant. commissure and left cord	Squamous cell carcinoma June 22, 1929	June 18, 1929 Loc. Nov. & Coc.	Perichondritis	Recurrence at 9 months	Atresia of larynx Death	2 years and 3 months
20.	Male, 63 yrs. Salesman	Hoarseness 1 year	Anterior two-thirds left cord	Squamous cell carcinoma June 22, 1929	June 26, 1929 Loc. Nov. & Coc.	2 weeks	None	Voice good Airway normal	2 years and 3 months

THYROTOMY OR LARYNGOFISSURE IN THE CURE OF EARLY INTRINSIC CANCER OF THE LARYNX.—(Cont.)

Series No.	Age and Sex Occupation	Duration of Symptoms	Location of Lesion	Date of Biopsy	Thyrotomy date Anesthesia	Convalescence Complications	Recurrence Metastasis	Result	Period of cure, to Sept. 1931
21.	Fem., 52 yrs. Housewife	Hoarseness 18 months (intermittent)	Anterior commissure right cord	Papilloma beg. cancer July 30, 1929	Aug. 26, 1929 Loc. Nov. & Coc.	10 days	None	Voice good Airway normal	2 years and 1 month
22.	Male, 48 yrs. Prof. Golfer	Hoarseness 1 year	Mid-two-thirds right cord	Squamous carcinoma	Sept. 26, 1929 Loc. Nov. & Coc.	10 days	None	Hoarse whisper	2 years
23.	Male, 63 yrs. Manager	Hoarseness 4 months	Anterior two-thirds right cord	Early squam. epithelioma Dec. 10, 1929	Dec. 17, 1929 Loc. Nov. & Coc.	10 days	None	Whisper Glottis normal	1 year and 9 months
24.	Male, 68 yrs. Gen'l Mgr.	Hoarseness 3 months	Anterior end left cord	Squamous cell carcinoma May 29, 1930	June 2, 1930 Loc. Nov. & Coc.	3 weeks granuloma	None	Good whispered voice	1 year and 3 months
25.	Male, 65 yrs. Ins. Agent	Hoarseness 1 year	Left cord	Squamous epithelioma May 3, 1929	Nov. 23, 1930 Loc. novocaine	None	Recurrence Partial laryngectomy Laryngectomy	No metastasis Larynx removed	
26.	Fem., 26 yrs. Teacher	Hoarseness 2 years	Mid-third left cord	Squamous carcinoma Dec. 30, 1930	Jan. 17, 1931 Loc. Nov. & Coc.	2 weeks granuloma	None	Good whispered voice	9 months
27.	Male, 60 yrs. Salesman	Hoarseness 1 year	Anterior end right cord	Squamous epithelioma Feb. 17, 1931	March 9, 1931 Loc. Nov. & Coc.	3 weeks	None	Good voice Normal airway	6 months
28.	Male, 48 yrs. Cotton Buyer	Hoarseness 9 months	Anterior end left cord	Papilloma beg. cancer June 24, 1931	June 27, 1931 Loc. Nov. & Coc.	2 weeks	None	Good voice Normal airway	2 months
29.	Male, 65 yrs. None	Hoarseness 4 months	Anterior 3rd right cord	Cancer June 18, 1931	July 1, 1931 Loc. Nov. & Coc.	3 weeks	None	Good voice Normal airway	2 months
30.	Male, 72 yrs. Text. Carder	Hoarseness 2 months	Anterior end left cord	Squamous cell epithelioma Aug. 25, 1931	Sept. 3, 1931 Loc. Nov. & Coc.	2 weeks	None	Good voice Normal airway	Recent

DISCUSSION.

Dr. Gordon B. New, Rochester, Minnesota: Dr. Tucker has given us an exceedingly good presentation of the diagnosis and treatment of early carcinoma of the larynx. In the very early cases that he refers to, I think a cure should be expected. As a rule, patients with cancer of the larynx, as in cancer elsewhere, have but one chance to get well if operated upon I believe that Dr. Tucker is a little optimistic about the end results of laryngectomy following recurrence in these cases, although in the low grade carcinomas one may do a laryngectomy where there is a recurrence following laryngofissure, and the patient will remain free of recurrence for many years. However, in many of these tumors the recurrence may not only be local but also metastasize to the glands of the neck, so that the chance of a good result from surgery is not good.

I believe the success or failure of any operative treatment of cancer of the larynx usually depends more upon the type or grade of growth present than upon any particular technique employed in the treatment of the tumor. Fresh frozen sections should be made just before the surgery is done, in order to determine the grade of malignancy present. This is very essential in deciding on the best possible treatment in the individual cases. In patient with carcinoma, graded four, of the larynx, unless the lesion is very small a laryngectomy should be done. A large, low grade tumor may be removed in a conservative way with good results, while with a tumor of similar size and of a high grade of malignancy the results with a conservative operation would be very poor.

In Colledge's recent report of the microscopic examination in Sir St. Clair Thomson's cases, he found that there were 25 per cent recurrences in the entire series of 60 cases. Of the high grade tumors there were 72 per cent recurrences, and it is in this group that we feel a laryngectomy should be done rather than a conservative operation. Of the low grade tumors there were only 6.9 per cent recurrences.

As to the question of technique, we prefer a two-stage operation in cases of indeterminate extent, and in cases where it is impossible to tell before the operation whether laryngofissure or laryngectomy should be done. This is particularly true in cases that have had a biopsy done previous to coming for examination. In many of these cases there is slight fixation, and it is difficult to tell whether this is the result of reaction from the biopsy or whether it is due to the carcinoma. In this group of cases we prefer a two-stage operation, first freezing up the neck, as one would at the time of a first stage laryngectomy, and then at the time of the second stage a laryngofissure is done. If it is possible to remove the growth in a conservative way with or without removal of the cartilage this procedure is followed. If this is not thought advisable then a laryngectomy can be done with no added risk.

We use Nembutal as a preliminary medication the night before the operation and the morning of the operation. This allows the patient to come to the operating room in the best possible condition. Following operation they wake up soon so that they are able to cough up their tracheal secretions.

In patients with short necks we divide the hyoid bone in doing a laryngofissure, as we believe this allows the larynx to come up into the wound and gives much better exposure. We also believe that surgical diathermy used in cases in which laryngofissure is done is a distinct advantage. If one understands the diathermy apparatus that is used there is no need to get extensive sloughing, as has been reported by some.

In the last six years we have operated upon forty-one cases of early epithelioma of the larynx by means of laryngofissure, and during this time we have had 13 per cent recurrences, the patients being well from three to six years.

Dr. Robert Sonnenschein, Chicago: This morning I had the pleasure of reading Dr. Tucker's paper, which is notable for its clarity and precision.

My partner, Dr. Pearlman, and myself have during the past three or four years been doing laryngofissure or hemilaryngectomy at times after the method of Dr. New, and at other times following the method of Sir St. Clair Thomson, and thus far we have had favorable results. As a rule we use amytal, six grains, one hour before operation, then use novocain at operation, and apply cocaine when the larynx is opened. We find dividing the hyoid bone is a good idea.

I think it is sufficient to say that the most important points which have been stressed by Dr. Tucker are careful examination, early diagnosis, and then employment of the operator's judgment in deciding the type of operation and the extent of operation which will best serve the patient.

Dr. H. L. Pollock, Chicago: Do you not in a great many cases get a large amount of granulation tissue without packing? I have been in the habit of doing a tracheotomy and packing to get rid of granulations.

Dr. Gabriel Tucker: Replying to Dr. Pollock, possibly 40 per cent of these cases had granuloma. The case is always examined to see whether or not that occurs.

Dr. H. L. Pollock, Chicago: In these cases you heard Dr. Tucker say he removed the granulations afterwards. When I was in Vienna I saw many of these cases, and they do a tracheotomy first and then pack the larynx for about a week. I have been in the habit of doing that and there are no granulations and no secondary operation afterwards. In many of these cases there seems to be a regeneration of the cord. On looking in the larynx afterwards you see almost an exact duplication of the cord, which I think is due to the fact that the granulations are not disturbed and removed secondarily. In a series of cases I did not use the technique described here today, but exposed the thyroid cartilage and made a window through it, which affords a better view, and it heals quickly. Then you do not have the danger of perichondritis, which often happens when you attempt to stretch the larynx open. The preliminary tracheotomy with packing in my hands is much better than the one-stage operation.

Dr. T. E. Carmody, Denver, Colorado: Dr. Tucker has covered this subject so well that, with the discussion of Dr. New and Dr. Sonnenschein, it is like carrying coals to Newcastle to say anything.

One of the points Dr. Tucker made was vocal rest in these cases when you want to take time to examine them—a day or two. It is not necessary to let them rest longer, because the diagnosis should be made at once.

I think among the younger men the laryngeal mirror is not used as much as it should be; and perhaps the direct speculum should be used more.

Many of the cases we have seen have had silver nitrate used, not only in cases of carcinoma, but in tuberculosis, and have been irritated a great deal. In our experience the use of an endothermic knife has been valuable, not only for the removal of specimens at the operation, but also for melting down granulations afterwards.

Dr. Joseph C. Beck, Chicago: The subject has been so well covered that I do not feel I should get up except to say that I, too, have done this work and failed so many times that I wish I were young again and could start all over. I failed because primarily the cases came too late. That is one of the points stressed in this paper—early diagnosis. Those who go around the country should impress it on the laryngologist in the small community who does not want to take the risk either to operate or to make a biopsy, that delay is invariably fatal. Because of his not being well informed as to the differentiation of tissue in the various types of epithelial growth, he allows valuable time to elapse, so that a laryngectomy must be done and the patient has to wear an artificial larynx or depend upon bucco-esophageal voice. For a man who has to depend upon speaking for his living, the laryngectomy operation, while it is a cure, is not to be compared with a laryngofissure. An operation such as described in this paper, and as most men are doing now, I think is what we may look forward to.

The endothermic knife when fully developed, I think, will be very valuable in these cases, preventing any possibility of spreading or implantation of cells, as Dr. Bloodgood has shown. I have quoted him a good many times to my classes on the question of "How long does cancer remain cured?" He had a case of cancer of the breast which after having been cured for twenty years recurred. So the time limit is not to be set at five years or any number of years.

I would like to ask Dr. Tucker about one point, that is, that with the Jackson scissors it is not always possible to cut through the thyroid cartilage because, as Dr. Pollock has recently shown in the review of an article by Prof. Oscar Meyer of Vienna, that there is not always calcification present but true bone formation in many instances, and in trying to cut through in a laryngotomy, I also found it necessary to use a saw to get through this tissue. We personally have had several such experiences and find that a single circular electric saw devised by associate, Dr. M. R. Guttman, which is about one inch in diameter, is of great value. This instrument will be described in the literature at some future time.

Dr. Gabriel Tucker, closing: I wish to thank Drs. New, Sonnenschein, Carmody, Pollock and Beck for their discussion.

In the two cases of recurrence in this series, the growth was within the larynx. In the first case no evidence of metastasis was found, but

the patient refused laryngectomy. In the second, a laryngectomy has been done and no evidence of metastasis outside the larynx was found. I believe that if recurrence takes place it will be first locally in the cases where the involvement was in the "anterior intrinsic area," especially if the cartilage is not removed at laryngofissure. This being the case, the patient has a chance to save the larynx and still a laryngectomy will be effective in cure if recurrence takes place, which it may in a very small percentage of cases.

The grading of the type of cancer is very important as Dr. New suggests. The type four lesion in the posterior portion of the larynx should have a laryngectomy immediately, but the same type of cancer in a small lesion in the anterior intrinsic area may be operated by laryngofissure thereby giving the patient a chance to save his larynx. I think that the type one and two cancer in the posterior portion of the larynx should have a laryngectomy also. I do not agree with Dr. New that the type of cancer is more important than the location and extent of the lesion.

By the Jackson technique, without cutting the hyoid bone, a sufficient exposure can be obtained of the interior of the larynx if the case is one suitable for laryngofissure. If the lesion is too extensive and posterior, Sir St. Clair Thomson's technique with the removal of the cartilage will permit a complete evisceration of the larynx. This can be used in those cases where laryngectomy can not be done, and radium can then be applied in the larynx if it is desired. I see no advantage to the two-stage operation because if there is evidence of metastasis, laryngofissure should not be done and laryngectomy is the patient's only chance.

Dr. Sonnenschein mentions the use of amytal. It has been my custom, unless the patient is very apprehensive, not to give a sedative prior to operation. If the patient is very nervous, morphine and atropine are given an hour before the operation and the laryngofissure is then completed under local anesthesia. Morphine and all cough sedatives are withheld after operation. Great care is used in carrying out this rule.

In answer to Dr. Pollock, masses of granulation tissue occur in about 40 per cent of the cases. We remove these by direct laryngoscopy without anesthesia. It requires only a few minutes and usually one removal is all that is required. This tissue is submitted to histologic examination. My experience with the pack in the larynx where it was necessary to use it has been that these cases are more likely to develop a perichondritis and excessive granulations than the ones where the larynx was closed. I have found that the best way to prevent perichondritis and granulation formation in the larynx is to suture the skin and subcutaneous tissue over the larynx without a pack as shown in the demonstration of technique.

Dr. Carmody has well said that we should not wait too long in making the diagnosis, but occasionally we find a patient, a case of "chronic hoarseness" where at first the lesion is not positive for cancer. A few weeks' observation in these cases, I would feel, is permissible until the lesion can be proved to be cancer clinically and by biopsy. The use of the endotherm knife where the cartilage is not removed does not seem necessary or advisable because we can get along very well without it and there

is danger of producing a chondritis. If the cartilage is removed at the time of laryngofissure I would see no objection to the use of the endotherm knife.

Dr. Beck's question, "When is cancer cured?" is very important and very difficult to answer, but from the fact that cancer may occur coincidentally in two locations of the body where the types of cell can be demonstrated to be different histologically and there is no probability of metastasis, why could it not be possible for cancer to occur again after a complete removal in the form of a new growth rather than a recurrence? In answer to Dr. Beck's other question regarding calcification and ossification of the thyroid cartilage, in a number of cases we have found this to occur. If it is present, we do not use the turbinatome, but use the Clerf or Gigli saw to split the thyroid.

In closing, I should like to emphasize again the great importance of a consideration of the location and extent of the lesion in the decision as to the advisability of attempted cure by laryngofissure in the particular case.

REPORT UPON AN EXPERIMENTAL INVESTIGATION OF THE EQUILIBRIAL MECHANISM OF THE FROG'S LABYRINTH.

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Montreal.

The present research was carried out in conjunction with Professor John Tait of the Department of Physiology, McGill University, and it is with his consent that the following advanced report is being prepared for the Research Committee of the American Academy of Ophthalmology and Otolaryngology. A more detailed report of these experiments will appear shortly in one of the journals of physiology.

During the past two years we have perfected our methods of operating upon the labyrinth of the frog, with the result that our lesions are more selective and more extensive. There are many reasons for the improvement: we are becoming more familiar with the structure of the labyrinth and have devised better methods of approaching the intralabyrinthine structures. We have improved our instruments, but what has contributed most to the advancement is the fact that now we operate with a constant stream of frog's Ringer solution flowing over the wound during the whole time of the operation. For this idea we are indebted to Professor von Frisch of Munich. The fluid serves the double purpose of washing away the blood from any of the smaller vessels which might be oozing, and it also washes away the bone dust from the dental drill which we now use for all approaches to the interior of the labyrinth. We also feel that our findings are now more valuable because we have developed additional methods of examination for labyrinthine lesions.

Formerly we were unable to affect lesions of the utricle without at the same time injuring the adjacent structures, whereas now

*I wish to express my indebtedness to the American Academy of Ophthalmology and Otolaryngology for the monetary grant made to me as Research Fellow of the Academy in Otolaryngology, part of which was used to defray the expenses of this research.

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we can with a high degree of certainty eliminate the utricle by detaching its nerve from the macula on one or both sides of the head.

We have given up the method of cauterizing the ampulla in order to eliminate a semicircular canal, because we have found that with better instruments it is now possible to separate the nerve just as it becomes attached to the ampulla without injury to any of the other structures, and there is no pulling on any other nerves. When using the cautery it was a common occurrence to find that, for instance, the anterior vertical and horizontal ampullæ were both involved in a lesion which had been designed to destroy one of these ampullæ.

By severing the nerves to the different ampullæ, to the saccule, to the lagena and the utricle, it has proved possible to have isolated lesions for study, or any combination of lesions which we desire. We have, therefore, been able to check our observations rather elaborately with the results to be described below.

We have confined our observations to the influence of the labyrinth on the body and limb musculature rather than upon the eyes, and we have made a special effort to determine just what part the labyrinth plays in everyday life, with special reference to the animal's movements and postures.

During the past two years we have repeated all the different combinations of lesions which we have previously reported from time to time, and it is gratifying to note that the majority of our observations were accurate so far as they went. In many instances we have recorded phenomena which at the time we could not understand and which in some cases were contrary to our preconceived ideas, but when reviewed now in the light of present knowledge, become quite clear and are in line with the rest of the phenomena.

The operations on the nerves have been checked by postmortem examination with osmic acid or by histological section of the whole head to compare both labyrinths. Some of these slides will be included in the report to appear in the near future.

In the present report I shall try to avoid unnecessary detail and shall just give a general outline of what we believe is the actual mode of working of the frog's labyrinth. The fundamentals herein outlined will probably be found to hold good throughout the vertebrate phylum but undergoing a great deal of

modification in different species, depending upon the needs and the subsequent development of the animal in question.

THE NORMAL REACTION TO ROTATION IN THE HORIZONTAL PLANE ABOUT A VERTICAL AXIS.

The Parts of the Labyrinth Concerned.—This necessitates a normal functioning labyrinth in all details. It is ordinarily considered that the horizontal canals are the parts of the labyrinth solely concerned in this reaction, but this is only partly true.

The Normal Reaction.—It has been many times described; the animal will usually walk around in the opposite direction to the turning, supposedly making an effort to retain the original field of vision. Sometimes, however, the frog will only turn the head in the opposite direction to the rotation, but will not walk around. On cessation of the rotation, especially if the stopping is sudden, the frog may then turn in the opposite direction to that in which it had been going—i. e., it now turns in the direction in which the table had been rotating. Instead of actually walking around on cessation, the frog may only swing the head around in the opposite direction to that in which it was held during the turning —e. g., while rotating to the right the frog will walk around or simply turn the head to the left, and on cessation of the rotation the frog will walk around to the right or simply turn its head to the right. What must be stressed is that during these reactions the frog's head is held steadily in the horizontal plane without any dipping or swaying, and all the limb movements necessary for turning, though alternate, are perfectly executed and synchronized. It is interesting to note the part played in this reaction by receptors other than the horizontal canals.

Removal of the Horizontal Canals.—If one or both of the frog's horizontal canals are removed and the eyes are not shielded there may be a reaction to rotation closely simulating the normal. If the eyes are shielded there is no reaction to turning to the side of the removed canal (by removing a canal, we mean to remove it functionally by severing the nerve to its ampulla). On cessation of rotation there might be a slight reaction, owing to the reversal of the direction of the stimulation which is now affecting the opposite intact canal. Rotation to the side of the intact canal elicits a normal response in every respect—i. e., all the movements are normally executed; there is, however, no reaction on stopping the rotation. When both horizontal canals are absent and the

animal is shielded against visual stimulation, the frog is not affected by the rotation but maintains its original resting position during rotation to either side.

Removal of the Vertical Canals.—If the horizontal canals are intact and an anterior vertical canal of one side is eliminated by nerve section the frog reacts to rotation to either side. The reaction has been described by us in the past as normal, but on more careful observation and in view of other observations, it can be seen that in response to rotation to the non-operated side, when the animal is turning around in the opposite direction to the rotation of the table there is a tendency during circling to stumble forward occasionally on the corner of the body corresponding to the absent anterior vertical canal. This stumbling is also present on turning to the operated side but is less marked.

If a posterior vertical canal is absent and the horizontal canals are intact there is a tendency during circling for the frog to sway backwards towards the corner of the body corresponding to the absent canal. The disability is more marked when the frog is turned towards the non-operated side.

If any two vertical canals are absent, this tendency to stumble is more marked, and if any diagonal pair are absent, or if all four vertical canals are absent, the stumbling becomes a veritable swaying to and fro as the animal attempts to walk around in the opposite direction to the rotation. The head is no longer held constantly level during turning.

Removal of the Utricles.—If the utricle is removed on one side the animal takes up the position described and illustrated by Ewald as typical of a unilateral labyrinthectomy in the frog. There is a marked dip of the head to the side of the operation, with flexion of the arm and leg on the operated side and extension of the arm and leg on the non-operated side. On the horizontal turn-table, rotation to the non-operated side accentuates the corkscrew-like twist of the animal to the operated side with a raising of the head on the opposite side and an increase of extension in the arm of that side. Rotation to the operated side brings about a partial undoing of the corkscrew-like twist to the operated side, the head being brought down more to the horizontal plane, the arm on the side of the lesion being more extended and the arm on the opposite side being more flexed. On cessation of rotation the usual unilateral labyrinthectomy position is again assumed.

If both utricles are removed the animal is symmetrical while at rest. When rotated on the horizontal turn-table it leans down to the side away from which it is being rotated. This lean is maintained during the whole period of rotation. Even if the animal walks around in the opposite direction (which it seems very loath to do in the absence of utricles) the lean is maintained. In other words, in the absence of the utricles the head is no longer kept level by all six semicircular canals during simple rotation about a vertical axis.

Removal of Horizontal Canals and Utricles.—If both utricles and both horizontal canals are removed, the frog still shows some response to rotation on the horizontal turn-table. It lowers the side of the head and body on the opposite side to which it is being rotated—e. g., if rotated to the right, the left side of the head and body is lowered, there being a flexion of the limbs on the left side and a partial extension of the limbs on the right side, most marked in the fore limbs. This reaction must come from stimulation of the vertical canals. There is no tendency to turn the head away from direction of the rotation or to walk in the opposite direction to the rotation when the eyes are shielded—these being the reactions which are brought about in the normal animal by the horizontal canals.

Removal of Horizontal and Anterior Vertical Canals and Utricles.—This animal gives a definite reaction to rotation on the horizontal turn-table even when the eyes are shielded. If the table is rotated to the right the right leg is extended, or possibly both legs will be extended, the right usually first; the right arm is partially extended and the left arm is flexed so that the left side of the head dips down and there is a corkscrew turning of the body to the left on its long axis. The reverse of these procedures is true on rotation to the left. With careful manipulation such an animal can be made to rest quietly on its back on the horizontal turn-table. In such a position it usually keeps all four limbs flexed. The slightest rough handling will cause the animal to right itself. If rotated to its right the most obvious reaction is a rapid throwing out of the right leg and a flexing of the left side of the head on the abdomen with a corkscrew-like turning about the long axis of its body so that it turns over into the normal squatting position with the right side of the body lowermost during the turning.

These latter reactions must arise from the posterior vertical canals because the remaining parts of the vestibular labyrinth have been removed. When one of the remaining posterior vertical canals is removed the reaction still persists on rotating to the side of the intact canal, but there is no reaction on turning to the opposite side. This is definite evidence that a vertical canal may be stimulated during rotation in the horizontal plane about a vertical axis.

It is interesting to note that this reaction just described, which results from stimulating the posterior vertical canal while the animal is on its back, is the normal response from a posterior vertical canal—i. e., extension of the leg on its own side and a dipping down of the head and flexion of the arm at the diagonal corner of the body. This would suggest that no matter in what manner a canal is stimulated it always calls forth the same kind of muscle response.

This posterior canal response on the horizontal turn-table will also explain the partial undoing of the characteristic unilateral utricular pose during turning to the operated side on the turn-table which we have just described, and the increased inclination on turning to the non-operated side. In each case the posterior vertical canal is stimulated by turning to its own side—e. g., if it is a right utricular lesion the frog leans down to the right, and if rotated on the horizontal turn-table to the right the lean is partly undone, because stimulation of the right posterior vertical canal causes an extension of the right leg and a flexion of the left arm with a dipping down of the head on the left side, which tends to overcome or correct the utricular inclination to the right.

Removal of the Horizontal and Posterior Vertical Canals and Utricles.—To date we have not succeeded in detecting a response from the anterior vertical canals when subjected to rotation on the horizontal turn-table about a vertical axis when all the other parts of the vestibular labyrinth have been removed.

Summary.—It has been pointed out that in the frog if any of the vertical canals are removed (the utricles being intact) there is a tendency for the animal to stumble in the plane of the absent canal when reacting to rotation on the horizontal turn-table. Even though the head is maintained in the horizontal plane for the most part there are these momentary departures from the horizontal owing to the absence of the quick corrective action

of the removed vertical canal. If a pair of canals, or more particularly, a diagonal pair of canals, are absent the departures become a veritable sway backwards and forwards in the plane of the absent canals. If both utricles are removed in a frog and it is rotated on the horizontal turn-table there is a permanent dip of the head during the rotation down to the side opposite to which it is being rotated.

The utricles overcome any gross or sustained effort to move the head out of the horizontal plane, the more sensitive vertical canals overcoming any slight tendency to move the head from dead level which might arise during rapid movement. The vertical canals and the utricles exert a checking influence upon one another, the net result of which is to maintain the head in the horizontal plane while the horizontal canals bring about the counter turning reactions of the head and eyes and body destined to maintain the animal's original field of vision against any tendency to rotate the frog about a vertical axis.

SPONTANEOUS MOVEMENTS.

Forward Progression.—When a normal frog is placed on the floor it may remain quiet for a time, but as soon as its skin begins to dry it will hop off by taking long leaps in some particular direction. When examining our operated frogs while they were free on the floor, it was noted that some of our animals would hop off, even though there may have been some imperfection in the take-off or in the landing, while others would just circle around and never leave the same small area; in other words, in some of our operated frogs something had interfered with their forward progression. Frogs that had lost either the horizontal canals or the utricles were the ones which had lost their power of forward progression in the majority of instances. If only one horizontal canal is removed the frog can still go forward, but there is a tendency to circle. A frog that has lost both horizontal canals or both utricles tends to circle on attempting any spontaneous movement. If the vertical canals are injured, the frog can still hop straight ahead, but there is usually some imperfection in the take-off or in the landing.

Decanaliculate Posture.—In our previous publications we stated that removal of a semicircular canal does not alter the posture of the frog when at rest. This statement was based on the fact that following a semicircular canal lesion there is no permanent

forced position, whereas after a lesion of one utricle there is a permanent forced position. However, after any canal lesion there may be a slight departure from the normal resting pose. This alteration in posture is most often seen just after a movement; in fact, it is usually a persistence of the posture in which the frog was during the latter part of the movement, and for this reason it might be called a "residual pose," or perhaps better a "decanaliculate posture." If a frog with a lesion of the right horizontal canal is circling to the right, on cessation of the circling it may come to rest with a slight lateral curvature of its back to the right. After a few moments it may, for no apparent reason, make a movement, straighten out and assume a normal pose. This curved posture is not a forced position, because the frog can adopt any other posture at will, but it is more likely to adopt this particular pose. There is a characteristic pose for each canal lesion, so much so that it is in many cases symptomatic of the canal lesion. If an operated frog is placed on the floor an experienced observer can name the semicircular canal lesion after watching a few spontaneous movements and the subsequent residual pose. With a lesion of one anterior vertical canal there is a tendency for the frog to come to rest with the operated side of the head slightly below the horizontal plane. If the lesion is of a posterior canal, for instance, the right, there is a tendency for the frog to come to rest with the left side of the head held upwards and backwards to the right above the horizontal plane. A frog with both anterior vertical canals removed tends, after movement, to sit with its head dipped slightly down. A frog with both posterior vertical canals removed tends to sit with the head slightly cocked up. Any of these poses may only be held for a few moments and they can be altered, so they are not to be confused with forced positions which result after unilateral utricular lesions. If a frog has a decanaliculate posture at the beginning of a movement or leap, the direction of the movement will be determined by the decanaliculate posture—e. g., if an animal with a lesion of the right horizontal canal has a lateral curvature of the vertebral column to the right and it jumps, it tends to deviate to the right in the air. A decanaliculate posture is permanent to the extent that it will persist throughout a movement and alter the course of that movement. The decanaliculate posture may be explained by the fact, to be brought out later, that the canals work in pairs, two physiological opposites in each plane,

and they are so regulated that they both elicit reflexes which oppose each other and tend to bring the head into the exact symmetrical normal posture. In the absence of one canal, the opposite canal tends to over-correct for any head movement in its plane, so that when the animal comes to rest, the corner or the side of the head corresponding to the intact canal is just a little above the horizontal plane or a little beyond the midline. The existence of decanaliculate posture shows that the utricle of itself will not necessarily maintain the head exactly level without the coöperation of all six canals.

THE NORMAL REACTION TO ROTATION IN A VERTICAL PLANE ABOUT A HORIZONTAL AXIS.

The Parts of the Labyrinth Concerned.—As a result of our previous investigations we had come to the conclusion that rotation about a horizontal axis stimulates the semicircular canals when the movement is rapid or sudden, and stimulates the utricles when the movement is slow. However, more careful observation of frogs with different combinations of canal and utricular lesions has necessitated a slight modification of this simple view, for the same close coöperation between the canals and the utricles is found as is the case during rotation in the horizontal plane about a vertical axis.

The "Chin-lift" and the "Chin-drop" Test.—These tests have contributed greatly to the improvement of our examination of the frogs. They are an alternative to slow and fast tipping on the tilt-table, but are much simpler of application and in many cases furnish information about the canals which is not so clearly demonstrated on the tilt-table. If the chin of a normal frog is raised by a sudden lift of the forefinger, the movement is resisted, and the animal retains pretty much the normal pose that it had previously. The same is true if the chin is gently raised and then is suddenly dropped by quickly withdrawing the finger. The abnormal responses elicited by the chin tests will be described as we discuss the various parts of the labyrinth concerned in rotation about a horizontal axis.

The Normal Reaction.—When an animal is rotated about a horizontal axis, it makes every effort to keep its head in the horizontal plane. If the rotation is slow, the counter reaction is also slow; but if the rotation is fast, the counter movement of the head is fast. The reaction is very smooth and very well timed, perfectly

executed and is never overdone, but the head is always kept level. The reaction is sometimes so fast that it is very difficult to follow the intermediate stages of the movement.

If the rotation of the table is down, the frog raises its head by extending the arms forward, the body being pushed back between the flexed thighs; if the tilting is to one side, the limbs on the side to which the table is tipped are extended, the opposite limbs being flexed so that the head is held level; if the tipping is diagonally forward—e. g., forward and to the left, the left arm is extended, the right arm is partially flexed, the left leg is extended forward, the right leg being flexed, and the body is pushed backwards and to the right; if the animal is tipped backwards the head is lowered till the snout touches the table, the arms are flexed and the elbows turned out, the legs may be partially extended and the animal may even walk up the board or table. The end stages of these reactions are well seen in Figs. 1 to 5, page 143, Tait and McNally, 1925.

Removal of the Posterior Vertical Canals.—After removal of both posterior vertical canals the frog no longer lowers the head promptly when tipped backwards, but, on the contrary, the head is thrown upwards and backwards, the arms are extended and the whole body moves backwards. These movements are relatively slow, and only when they are completed does the animal make the normal counter reactions to backward tipping, viz., lowering of the head to the table, flexing of the arms, outward spreading of the elbows and extension of the legs. This reaction is quite different from the reaction of a delabyrinthized frog to backward tipping. The latter frog makes little or no response to backward tipping, and is not easily thrown off the tilt-table, whereas, the frog, deprived simply of posterior vertical canals, is very easily thrown off the table backwards, because the first part of its reaction to backward tipping is in the wrong direction and if continued is destined to throw the frog from the tilt-table backwards.

If in addition to the two posterior vertical canals the two anterior vertical and the two horizontal canals are removed, the upward throwing of the head on backward tipping is still present, but the reaction disappears after the two utricles are removed. This upward throwing of the head to backward tipping, in the absence of the posterior vertical canals, must be in some way connected with the utricles.

In one of our first publications (McNally and Tait, 1925, page 165) this abnormal reaction is noted and commented upon, though we were at a loss to explain it. We state, "Here the peculiar fact should be noted that on backward tipping the frog makes a definite movement with its arms in the opposite sense to that of a frog with intact posterior vertical canals." The real significance of this reaction will be considered when we consider the utricle.

A Tetanic Reaction.—A further reaction might be noted here, though at present we can offer no explanation for it. When a frog without posterior vertical canals is tipped backwards, the head is raised and the arms are extended as has just been described above. Sometimes, however, just at the top of the movement the animal may develop what might be termed tetany; the arms become more rigidly extended, the back is curved backward, the legs are extended, the eyes are closed, and the whole body trembles with spasm which lasts for a second or two; as the spasm passes off, the animal then dips forward to assume the normal pose for the backward tipping. The tetanic reaction is more easily elicited at one time than it is at another, and if it appears once it can be made to reappear on several successive tippings. It has been noted only after lesions of the posterior vertical canals, and it may occur after a lesion of only one posterior canal, though it is more common after a lesion involving both canals.

The Chin-lift Test.—If the chin of a frog deprived of both posterior vertical canals is given a quick lift upwards with the finger, the head goes up easily and remains up; the arms having been extended during the upward movement keep the head from returning to its original position. If the weight of the head is supported on the finger and the finger is then suddenly withdrawn, the head does not drop but is supported by the outstretched arms. In other words, the head is very slack and is easily tilted upwards in the absence of the posterior vertical canals, but it resists forward tipping normally.

Decanaliculate Posture.—The frog deprived of posterior vertical canals shows some abnormality in jumping when placed on the floor. During a jump the head may be held high in the air and the animal lands more on the hind part of the body than usual. When it comes to rest, the head may be cocked up as described after the chin-lift test. This is the characteristic decanaliculate posture in the absence of both posterior vertical

canals. It is the same type of residual pose as was described for the horizontal canals. The intact anterior vertical canals have over-corrected the position of the head, so that it is held a little above the normal level. If this pose is present when the animal jumps, it tends to shoot up into the air, sometimes even turning a somersault backwards.

Removal of the Anterior Vertical Canals.—If such a frog is tipped forward, the head dips down, the body moves forward between the loosely flexed thighs, and it is only towards the end of the tipping that the head is slowly raised, the arms being extended forwards in an attempt to push the body backwards between the thighs. The abnormal forward slewing in this frog is not such a striking phenomenon as the backward slewing just described which takes place during backward tipping in the absence of the posterior canals, nor have we ever noted anything akin to the tetany which may occur at the height of the backward slewing. However, the forward slew during forward tipping is none the less definite though less marked.

The Chin-drop Test.—If the chin is suddenly dropped from the supporting finger, the head sinks forward and the arms flex rather than support the head. The head may remain down when the animal comes to rest. This is the decanaliculate posture in the absence of the anterior vertical canals.

If only one anterior vertical canal is removed and the chin is suddenly dropped, the head sinks down on the side of the absent canal. This is one of our surest and simplest tests for diagnosing an anterior canal lesion.

If one of the posterior vertical canals is removed and the chin is suddenly raised, the head goes up and backwards on the side of the absent canal. If one attempts to push the head up and backwards to the side of the intact canal the effort is resisted and the frog always returns to the normal pose. The chin-lift test is here also our most valuable diagnostic aid.

Spontaneous Movements.—After removal of both anterior vertical canals the animal has difficulty in effecting a long leap. It does not raise itself as high as the normal frog, the arms tend to spread laterally and the legs are not properly tucked under the animal when it lands. In swimming this animal tends to dive to the bottom of the tank. The anterior vertical canals are essential to keep the head raised under normal conditions.

Removal of Horizontal and Anterior Vertical Canals and Utricles.—In this frog all the vestibular structures are removed except the two posterior vertical canals. When tipped backwards there is a very rapid thrusting out of the legs, lateral extension of the arms with the palms turned backwards and lowering of the head. It is a very sudden reaction which takes place at the very beginning of the tipping. Sometimes the reaction is so brisk that the legs seem to be extended several times and the frog may wriggle off the table backwards. It is a more rapid and more extreme reaction than that of a normal frog. The chin-lift test also brings out this reaction—in fact, the frog gives the reaction so promptly that it is very difficult to get the finger under the chin. This reaction of backward tipping reminds one of the efforts that a swimmer, swimming breast-stroke, might make on wishing suddenly to swim to the bottom, because the whole movement seems to be designed to drive the animal downwards and forwards.

When tipped forward this animal pushes itself forward on the table with arms and legs extended, but there is no actual slewing forward of the body on flexed limbs nor is the head raised afterwards as when the utricles are intact.

A Combination of Decanaliculate and Deutriculate Posture.—This animal with only the posterior vertical canals intact may exhibit an extreme residual pose, because deutriculate posture is always more extreme than decanaliculate posture. Because the anterior vertical canals are absent the head is held below the normal level. If the animal is made to sit near the edge of a table it is possible, by gently applying the chin-drop test, to have the animal sitting with its chin over the edge of the table and dipping away below the surface as though the frog were gazing into the bottom of a precipice. If the utricles were intact, such an extreme posture would not be possible. A very striking proof that this posture is due to the overaction of the intact posterior vertical canals is the fact that if the posterior vertical canals are removed in this animal all suggestion of the head-down residual pose is permanently eliminated.

Removal of the Horizontal and Posterior Vertical Canals and the Utricles.—In this frog only the anterior vertical canals are intact. When tilted forward the head is raised, the arms are extended forward, the body being forced back between the flexed

thighs. This reaction might be slightly more exaggerated than the response of a normal frog, but there is nothing suggestive of the same degree of exaggeration as is shown by the frog with only the posterior canals intact when it is tipped backwards.

When the frog with only the anterior canals intact has its chin suddenly lifted, the head goes up and is maintained in a cocked-up attitude on the extended arms, there being an extreme residual pose of the type characteristic of the absence of the posterior vertical canals.

Labyrinthine Check Mechanism.—The exaggerated residual pose in these two animals and the extreme reaction to backward tipping of the frog with only the posterior vertical canals intact give further proof that in the normal animal the various parts of the labyrinth exert a check on each other, so that the reactions are never overdone and are graduated sufficiently to counteract the exciting stimulus. This check mechanism has already been suggested by the existence of decanaliculate posture following a canal lesion.

Removal of a Diagonal Pair of Vertical Canals.—If one anterior vertical and the opposite posterior vertical canal are removed in the frog, the animal's movements are disturbed. When tilted in the plane of the intact canals the reactions are normal. When tilted in the plane of the absent canals there is marked swaying. If the right anterior vertical and the left posterior vertical canals are removed and the frog is tilted forward and to the right, the head dips down and to the right, and this is followed by a wide swing backward and to the left above the horizontal as the table comes to rest. The animal continues to make oscillatory movements in this plane of less and less amplitude till it finally comes to rest with the right hand corner of the head either above or below the normal level of the head. In this animal the decanaliculate posture may be characteristic of the absent right anterior or of the left posterior vertical canal.

Spontaneous Movements.—When this frog attempts any spontaneous movement on the floor, or more particularly, if in shallow water, the oscillatory movements to and fro in the plane of the absent canals are very marked and very seriously interfere with the frog's progression. When the animal comes to rest after any movement there may be as many as five or six oscillatory movements before the animal can finally settle down. The chin tests

show that the head is very flaccid in the plane of the absent canals. If the chin is suddenly raised the head goes up and to the left, or if the chin is dropped suddenly the head goes down and to the right.

Watching this frog impresses one with the fact that the loss of a diagonal pair of canals is a much more serious handicap to the frog than is the loss of either the anterior, posterior or lateral pair of canals.

Removal of the Four Vertical Canals.—This frog has intact the two horizontal canals and the two utricles. The swaying to and fro is very much more marked than it is in the frog with a diagonal pair of canals removed, because in this animal both diagonal pairs of vertical canals are gone; therefore, there is no corrective mechanism at any corner of the body. The swaying may occur either backwards and forwards, or in a diagonal vertical plane about a horizontal axis (there is no side to side sway about a vertical axis because this is prevented by the intact horizontal canals). On the tilt-table the reactions are typical for a lesion of both the posterior and anterior vertical canals, viz., when tipped backwards there is the abnormal throwing upwards and backwards of the head with the extension of the arms, etc., which is followed by the tetanic reaction. In fact, it was in this animal that we first noted the tetanic reaction. When tipped forwards the head slews forward between the flexed arms as was described for the animal with only the anterior vertical canals removed.

The chin-lift and chin-drop tests bring out the different canal lesions very clearly. A very marked flaccidity of the head is noted in this frog. If the snout is gently grasped between the thumb and forefinger it can be moved up and down through a considerable angle without meeting any resistance. When the head is thrown up by a sudden lift of the finger it goes up and back, but it does not stay up as when only the posterior vertical canals are removed; it falls down between the loosely flexed arms, and it will move up and down several times before coming to rest. In the resting position the head may be a little above or a little below the normal head level because this frog may adopt a decanaliculate pose characteristic of the absent posterior vertical or of the absent anterior vertical canals. The flaccidity of the head suggests that the semicircular canals normally play some part in regulating the amount of tonus which is present in all

the muscles concerned in the maintenance of the head in its normal firm position, including the limb and neck muscles.

In swimming this frog rolls from side to side about its longitudinal axis, so that with alternate strokes of the limbs the white of the abdomen may show on alternate sides, but it will not turn completely over as will a frog after the utricles are removed. This animal's righting reaction is quite brisk.

The marked oscillations displayed by this frog on the tilt-table are even more marked when the animal attempts any spontaneous movement. If it is forced to leap when it lands, there is a very marked swaying in every direction before the animal finally comes to rest. One is impressed by the serious inconvenience it is for the frog to be without its four vertical canals.

The oscillations may be partly due to the absence of the canal check mechanism on each other, as shown by the presence of decanaliculate posture which the animal may adopt, but the most important factor is the absence of the check mechanism which the canals normally exert upon the utricles, because this swaying is not present if the utricles are removed. This will be explained more fully when we are discussing the utricle.

Removal of the Horizontal Canals and Utricles.—In this frog the four vertical canals are the only parts of the vestibular mechanism intact, so that it is the exact opposite to the last frog described. The contrast is interesting. In the frog with only the vertical canals intact one is struck by the steadiness of the animal. Careful observation reveals that there is a side to side sway about a vertical axis due to the absence of the horizontal canals, but it is only very slight and does not incommode the animal to anything like the same extent as did the swaying after removal of the vertical canals.

The reactions to quick tipping in any direction about a horizontal axis are normal in every respect. The animal's responses to slow tilting, if the tilting is carefully and smoothly done, are very poor, owing to the absence of the utricles. The animal may be tilted well out of the horizontal plane before it makes any corrective movements.

It has already been pointed out, when discussing rotation about a vertical axis, that this animal with the four vertical canals intact still gives a definite response to rotation in the horizontal plane, even though the horizontal canals are absent.

The responses to the chin-lift or chin-drop test are normal. The head is quite firmly fixed to the neck so that there is no flaccidity, as when the vertical canals are absent.

Spontaneous Movements.—This frog when placed on the floor shows a tendency to move around in the same small area; it is loath to jump, owing to the absence of horizontal canals and utricles, but if the animal is forced to jump, the landing is fairly normal. After coming to rest there may be a slight lateral curvature to either side, the decanaliculate posture for an absent horizontal canal.

When only the four vertical canals are intact the frog's spontaneous movements are curtailed, but one is impressed with the fact that it is much better able to resist stimulation than is a frog with horizontal canals and utricles intact and the vertical canals gone.

Direction of Stimulation of a Semicircular Canal.—Ewald (1892) has shown that a semicircular canal in the pigeon is stimulated by a movement of the endolymph either towards or away from the ampulla. He also found that a canal is maximally stimulated by a flow in only one direction and minimally by a flow in the opposite direction. The direction of maximum stimulation in a horizontal canal is a flow of endolymph towards the ampulla, and in a vertical canal the maximum stimulation is a flow of endolymph away from the ampulla. These facts, which were first discovered in the pigeon, are the basis for our clinical tests of labyrinthine function in man. However, it is generally believed that this same mechanism does not hold for all the different species of animals. We have previously pointed out, as has Maxwell (1923), that in the frog the horizontal canal responds to stimulation in only one direction. Our own observations have always led us to believe that this same rule holds true for the vertical canals in the frog, that they only respond to stimulation in one direction. The chin-lift and chin-drop tests furnish us with a more definite proof of this assumption. If a frog with only the posterior vertical canals intact has its chin lifted, the reaction of lowering the head and extending the legs comes about the very moment the head is even slightly raised. The reaction comes on at the very beginning of the lift. If the chin is very slowly raised and then suddenly dropped the reaction does not occur. The posterior vertical canals, therefore, are stimulated by the upward lift of the

head but not by a forward drop of the head. In terms of endolymph flow, this means that when the head is lifted suddenly there is a relative flow of endolymph in the opposite direction due to inertia of the fluid, which is a flow away from the ampulla in the posterior vertical canals. The sudden dropping of the head, which means a flow of endolymph towards the ampulla in the posterior vertical canal, does not elicit any reaction in the frog with only the posterior vertical canals intact.

If a frog with only the anterior vertical canals intact has the head thrown up suddenly by the finger, the animal makes no protective reaction: this is a flow of endolymph towards the ampulla in the anterior vertical canal, and it does not elicit a reaction. If the head is suddenly dropped, there is a prompt extension of the arms at the very beginning of the drop, so that the head is not allowed to fall: this means a flow of endolymph away from the ampulla of the anterior vertical canal and a very prompt response is elicited.

These two experiments would seem to confirm our previous observations that in the frog the vertical canals, as well as the horizontal canals, respond to a flow of endolymph in one direction only. For the vertical canals the direction of stimulation is when the ampullated end of the canal moves in front. The mechanism of stimulation of the canals in the frog is slightly different than in the pigeon or in man.

Summary.—When the normal frog is rotated about a horizontal axis it reacts in such a way as to keep the head always steadily in the horizontal plane. The reaction is not affected by removing the horizontal canals. If a vertical canal is removed and the frog is tipped in the plane of the missing canal and towards that corner of the body there is a definite slew in the wrong direction. If a pair of vertical canals is removed, the slew is more marked. If a diagonal pair of vertical canals is removed, the frog sways in the plane of the absent canals, backward and forward, to the slightest tipping; and if all four vertical canals are absent the swaying may be in any direction about a horizontal axis. If both utricles are removed in addition to the canals, the slew in the wrong direction and the swaying immediately disappear.

Normally, therefore, the vertical canals and the utricles are stimulated by rapid rotation about a horizontal axis, and the vertical canals must exert a checking influence upon the utricles at

the beginning of the stimulation, in order that the frog may carry out the normal reactions to the rotation so that the head remains level throughout the movement.

THE STATIC LABYRINTH.

The Saccule and the Lagena.—We have cut the nerves to the saccule and to the lagena on one and on both sides in the frog, and we have been unable to detect any change in the animal's vestibular reactions. These findings, as we have pointed out in previous publications, are confirmed by other observers. Apart from the experiment of Benjamins and Huizinga (1926), which indicates that in the pigeon the rotatory compensatory eye positions arise from the saccule, all other experimental evidence suggests that the saccule has no vestibular function.

The Utricle.—It is only within the past year that we have been able to effect clean operations upon the utricle. Prior to this we were obliged to infer the result of a utricular lesion when it was in conjunction with some other labyrinthine lesion. The difficulty has been that in the frog the utricular macula lies just over the conjoint nerve which supplies the anterior vertical and horizontal canals as well as the utricle. The fibers for the latter come off the upper surface of the nerve and immediately enter the under surface of the macula. It is, therefore, very difficult to separate these fibers without at the same time injuring the nerves going to the anterior vertical and the horizontal canals. However, we have succeeded. By modifying the approach and with the use of better and finer instruments we can at present be reasonably sure in doing an operation upon the utricle, that it is an isolated lesion, unless we choose to associate some other lesion with it.

Removal of the Utricles and Anterior Vertical and Horizontal Canals.—In this frog only the posterior vertical canals are intact. It was one of the first experiments in which we succeeded in cutting the conjoint nerve to the utricle and the anterior vertical and horizontal canals. One of the first things that impressed us about this frog was that it has a normal righting reaction when laid on its back. Because the righting reaction persisted when all six canals were removed, we came to the conclusion that it was a utricular phenomenon because it is not present in a delabyrinthized frog. If one of the remaining posterior vertical canals is removed in the frog under discussion, the righting reaction persists, and the righting may take place to either side, so that the

one remaining canal does not impose direction upon the righting reflex. The ability of the frog to right itself promptly is not solely dependent upon the utricles, and any part of the vestibular mechanism may initiate the impulse.

Maintenance of Posture.—With both conjoint nerves severed the animal can maintain any posture which is adopted. Posture is quite well maintained in the delabyrinthized frog, so that mere maintenance of posture is not the exclusive property of the utricle but is most likely a muscle phenomenon.

Unilateral Utricular Pose.—In a frog after a unilateral labyrinthectomy Beritoff (1928) noted that if such an animal is very carefully manipulated it is possible to correct the marked dip of the head to the operated side, so that the animal assumes and maintains a symmetrical pose of the whole body. The correcting hand must be withdrawn very carefully, otherwise the symmetry will not be maintained. Beritoff believed that some additional stimulation from non-labyrinthine receptors is necessary to make the frog resume its twisted posture. However, we have found that resumption depends upon a movement of the head. The ordinary movements of respiration are not sufficient to cause resumption, but, on the other hand, any slight jarring of the table or the very beginning of any spontaneous movement causes immediate resumption of the twisted posture so characteristic of a unilateral labyrinthectomy. The explanation would seem to be that while the animal is at rest and there is no movement of the head, simple pressure of the otolith upon the utricular macula is not sufficient to bring about resumption of the unilateral utricular position, but some little shift or accentuation of pressure must give the initiating stimulus to the utricle. This would suggest that normally the utricle is stimulated by some slight movement of its otolith, and it initiates the correct limb response, the new attitude being maintained by the muscles themselves, and that steady pressure or pulling of the otolith is not the adequate stimulus for the utricular macula.

Removal of the Utricles.—If only one utricle is removed the frog assumes a twisted position similar to the unilateral labyrinthectomy position. As soon as the remaining utricle is removed the animal again assumes a symmetrical position. When both utricles are removed and the six canals are intact, the animal shows very little disturbance to a casual observer. It is a little loath to jump but it lands quite well. During horizontal rotation

about a vertical axis the head dips out of the horizontal plane. When slowly tipped out of the horizontal plane the reactions are not quiet normal, but to quick tipping the reactions are quite brisk and normal. The animal is still able to right itself promptly on the ground, but in water it is apt to be a bit uncertain and may float on its back. On attempting any movement there is a decided tremor of the whole body. On the whole, it is surprising how relatively little disturbance is caused the frog by the loss of both its utricles.

Abnormal Utricular Reactions.—After removal of any pair of canals we have mentioned that the animal makes a definite movement in the wrong direction, and we pointed out that this wrong direction is unaffected by removal of the opposite pair of canals, but it is abolished by removal of the utricles. What happens may be explained as follows: When the animal is quickly tilted backwards, the utricular otolith is relatively thrown forwards by its inertia, if the posterior vertical canals are absent and the head is allowed to swing backwards. In a normal animal slow tilting forward of the frog, which should cause the otolith to exert pressure on the front of the macula, causes an extension of the arms and a raising of the head, but this is the abnormal reaction which occurs at the beginning of the backward tilt in the frog without posterior vertical canals, and must be due to the sliding forward of the utricular otolith on its macula. In the absence of posterior vertical canals after the first raising of the head and the extension of the arms to a backward tilt, the head is then lowered and the arms are flexed and the legs are extended. In the normal animal when it is slowly tilted backward so that its otolith slides to the back of its macula, the reaction is a lowering of the head, a flexion of the arms and an extension of the legs; but this is the reaction which follows after the first wrong reaction to the backward tilt in the frog without posterior vertical canals. In this frog, after the first forward movement of the otolith from inertia, towards the end of the tilt the otolith recovers and is carried to the back of the macula, when the reactions of lowering of the head, flexing of the arms, etc., are initiated. It would, therefore, seem that the utricle can well account for these apparently peculiar reactions which take place in the absence of the posterior vertical canals when the utricles are intact. The utricle will also explain the forward slew which occurs to forward tipping after the anterior vertical canals have been removed. The utricle will

also explain the oscillating movements which take place when all four vertical canals or a diagonal pair of vertical canals are removed, because once the head is allowed to swing out of the horizontal plane, the utricle develops a pendular motion of its otolith, which under normal circumstances with intact vertical canals cannot arise because the head is always kept level. It would seem, therefore, that the utricle is very susceptible to head motion about a horizontal axis and that the vertical semicircular canals guard against this by checking in the very beginning any slight tendency to head motion about a horizontal axis.

The two utricles oppose each other, so that their combined effect is to keep the head for the most part level, but they are not sufficient in themselves, as instanced by decanaliculate posture, and they require the checking influence of the vertical canals, as instanced by the oscillation movements in the absence of the four vertical canals.

Removal of All Six Semicircular Canals.—The only part of the vestibular mechanism which is intact in this frog are the two utricles. If tipped slowly about a horizontal axis it is able to make all the necessary protective reactions just as would a normal frog. This must mean that certain parts of the utricular macula must innervate the same limb and neck muscles as do certain of the canals, because the same type of reaction results from stimulation of either one; for instance, stimulation of the anterior part of the utricular macula elicits the same reaction as does stimulation of the anterior vertical canals, viz., the extension of the arms and the raising of the head, etc., to counteract forward tipping. The posterior part of the utricular macula supplies the same muscles as do the posterior vertical canals, viz., the muscles which lower the head, flex the arms and extend the legs to counteract backward tipping.

If the frog with only the utricles intact is tipped suddenly either forward of backward or to either side, it makes a wrong reaction, as we have mentioned previously, and the wrong reaction is due, as we have pointed out, to the inertia of the otolith of the utricular macula. It is interesting to note, on the other hand, that the canals are so constructed as to be maximally stimulated by the inertia of their contained endolymph and cupula following any sudden movement. From these remarks it would seem that the utricle is most unsuited to respond to any sudden stimulation. In the intact frog the canal response to any sudden tilt

is so rapid [the latent period of the response from a canal being only 35 as previously shown by us (Tait and McNally, 1929)] that the head is kept constantly level and therefore the utricle does not receive a very severe stimulus at the beginning of a sudden movement, or else the canal response is sufficiently strong to overcome any contrary reaction which might arise due to the inertia of the utricular otolith. The rapidity and exactness of the labyrinthine response in the normal frog to any sudden tipping would suggest that the utricular effect occurs only relatively late in the movement and determines the exactness of the new attitude to suit the new position of the animal, and the new attitude will be maintained by the muscles themselves until another movement occurs.

CONCLUSIONS.

1. Section of the nerve to the ampulla is the most satisfactory method of functionally removing a semicircular canal.

2. All parts of the vestibular labyrinth are concerned in the normal reactions of the frog to rotation about a vertical axis.

3. The vertical canals and the utricles are concerned in the frog's normal reaction to sudden tipping about a horizontal axis.

4. The horizontal canals bring about the counter turning reactions to rotation about a vertical axis which tend to retain the animal's original field of vision.

5. The vertical canals help to keep the head level during movement and if any one vertical canal is absent, there is an unsteadiness at that corner of the body corresponding to the absent canal.

6. The dipping of the head to one side in the absence of the utricles, during rotation about a vertical axis, is most likely brought about from stimulation of the posterior vertical canal on the side to which the frog is being rotated.

7. A posterior vertical canal always gives the same type of muscle response, whether it is being stimulated during rotation about a horizontal axis or during rotation about a vertical axis.

8. The vertical canals are stimulated during rotation about a horizontal or a vertical axis.

9. The horizontal canals and the utricles are evidently essential for forward progression in the frog, because in their absence the animal is condemned to shuffle about in a small area.

10. The semicircular canals are a factor in the maintenance of the normal posture of the frog. In the absence of any one canal

there is a slight alteration in the normal resting pose of the animal, which is characteristic for that canal, and which we have called "decanaliculate posture."

11. The semicircular canals influence the normal tonus of the neck muscles. In the absence of the canals the head is found to be quite flaccid.

12. The frog's righting reaction is independent of the utricle or of any other special part of the vestibular mechanism and is present so long as any one canal or utricle is intact to initiate the reaction.

13. A frog with one utricle removed can be made to assume a symmetrical posture under certain conditions. If the adequate stimulus of the utricle is simple pressure of its otolith this should not be possible.

14. In the absence of the vertical canals the utricles initiate wrong responses when the frog is tipped suddenly about a horizontal axis. Normally the canals must exert a checking influence upon the utricles.

15. The chin-lift and chin-drop tests are a great aid in diagnosis of a semicircular canal lesion in the frog.

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SYMPOSIUM ON AVIATION MEDICINE.

THE GOVERNMENT ASPECT OF AVIATION.

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The Government's interest in aviation dates back before the war but its interest in aviation medicine began during the war.

The hit-or-miss methods used early in the struggle, in the selection of flyers, having proved disastrous, more stringent regulations were adopted. The United States took a few cues from its allies, engaged in experimental work, observed its flyers and a method of selection and care of the flyer evolved.

This war method has gradually undergone further evolution based on practical experience and study. What seemed most important then has become least important today. What was slurred over hastily then has become emphasized today.

When civil aeronautics began to develop in this country, late in 1926, it became necessary to decide on regulations for selecting civil flyers. It became my duty as Medical Director of the Aeronautics Branch of the Department of Commerce, to devise these regulations. We had some military experience to go by, but no civil experience in this country. We had the experiences of other countries to help, but conditions vary so much in different lands that what will do for one will not necessarily do for another. Finally, however, a code of regulations was drawn up which was somewhat between the code of the International Commission for Air Navigation and the military regulations of this country.

It was recognized that civil flying differs from military flying in many respects, and that civil flying is of several classes.

After our regulations were adopted it became necessary to find qualified physicians to make the necessary examinations. There were at that time very few trained flight surgeons. The examination covered more ophthalmology than the average man doing general practice could handle without special instruction. The

ear, nose and throat which were paramount during the war, had been relegated to a very secondary place. Flying is no longer thought to be an ear problem. It is a problem of eyes and nervous system. The latter, therefore, involves a certain amount of psychiatry. Hence, our problem was solved in many places by appointing a team of two examiners, one to handle the general physical and the nervous system examinations; the other to handle the eye section, and incidentally, the ear, nose and throat.

I wish here to acknowledge the helpful co-operation of many of the members of this Academy who gave me material assistance in the early days by taking over these examinations when there was little likelihood of financial reward. The corps of examiners has grown now to about 800, but it was names like Berens, Fenton, Harkness and many more that persuaded others of the type desired to join in the work and keep it on a high plane.

I may say here in acknowledgment to both Assistant Secretaries of Commerce for Aeronautics that I was backed up to the limit by both Mr. MacCracken and Colonel Young in keeping politics out of the appointment of Medical Examiners. Not once was I forced to appoint a Medical Examiner merely because some politician wanted him appointed. His record was investigated and if he were not fitted for the post, he was not appointed, no matter how much political "drag" he had.

At the time I resigned as Medical Director, last November, over 100,000 physical examinations of pilots had been made by these examiners. Since then about 30,000 more have been made.

Transport and Limited Commercial pilots are examined twice a year; industrial, private and student pilots, once a year. There are now over 16,000 licensed pilots in this country today (exclusive of students). Of these about half are private pilots, about 1,700 limited commercial pilots and approximately 6,000 are transport pilots.

A study of the records of these pilots reveals that only about 7 per cent of our transport pilots have physical defects, even trifling in character. About 15 per cent of private pilots have them.

Over a year ago Cooper and I conducted a study of over 9,000 students. We have reported elsewhere the results of this study. Briefly, it showed that any deviation from the physical norm, no

matter how slight, interfered with the progress of the student in his flying. Of the physically defective (nondisqualifying defects) a percentage learned to fly that became increasingly smaller directly as the importance of the physical defects increased.

Cooper has continued this study and has only just reported that the study of approximately 30,000 students has confirmed our earlier work. He reports that of the physically perfect students 30.5 per cent were able to obtain a private license or better in one year. Of those with very minor defects, 17.5 per cent only were able to do as well. Of those with major defects (qualifying for private but not for transport) only 14 per cent could progress that far. Those unable to meet the private physical standard were physically disqualified for all grades. Yet there have been, all told, 286 men who were disqualified by the medical section who have been given a student permit. There were various reasons for their being given a chance. It is interesting to note that Cooper states only six of these have been able to progress far enough to obtain a license and hold on to it.

In other words, if you have any physical deviation from the normal, your chances of being able to learn to fly diminish in direct proportion to the severity of your defect.

This is not all. Cooper and I studied the effect of physical defect on the accident rate, and Cooper has recently continued that study on a group of about 4,200 accident cases. He found very definitely, as we both did on a smaller group, that the accident rate and the fatality rate among pilots with physical defects are far higher than among those who are physically normal. Dr. Berens has lantern slides to show statistically these facts, which are well shown.

He also classified these defects and, as would be expected, some defects are more serious than others. Minor defects of vision showed, for example, about 21 per cent of ability to progress as against 30.5 per cent for the normal. However, poor neuropsychic make-up and high blood pressure and pulse rates showed a rate of progress that was practically zero.

Bear in mind that by minor and major defects we mean defects that have not been considered serious enough to disqualify for a private license but only for a transport license and, in fact, some defects are so slight as not to disqualitfy for a trans-

port license. Yet, apparently, any deviation from the normal is a deterrent to flying ability.

It has already been stated that only approximately 30 per cent of students go on to a private license. Of course, some drop out for lack of finances, lack of time, etc. However, that a large percentage fall by the wayside because they are unable to learn to fly is borne out by the army's experience. They succeed in graduating only 46 per cent of their flying cadets.

Longacre studied the faculty reports on many of these rejections and found the reasons given by the instructor in recommending rejections were many, but there was a surprising frequency of the following: "Tenseness," "inability to relax," "unable to meet emergencies," "unable to correlate instruction," "forgets easily," "poor coördination," "clumsy," "slow in reacting," and so on.

These remarks suggest that a more careful examination of the nervous and psychological aspects would eliminate much unfit material. It requires training and persistence to get the necessary information. The past life must be reviewed in order to elicit the reactions to stress and emergencies. How has he handled difficult situations? An estimate must be made of his nervous stability.

The part played by the eyes, ears, nose and throat will be covered by Doctors Berens and Fenton, so I shall not go into those matters.

The Government wants the coöperation of the medical profession in keeping the pilot examinations of high standard and uniform in character. It wants only the highest type of physician, both professionally and personally, to make these examinations. It is interested in permitting all to fly who are physically and technically competent, and it wants to keep them in sound condition after selection. Safety in aeronautics—safety for the public, the passenger, the pilot and property—is its aim.

It is a distinct boost to aviation medicine that a body so distinguished as this has seen fit to recognize it as a specialty. Aviation is here to stay, and its medical problems have not all been solved. There is a very important field in supervising the flyer from a medical standpoint, into which I have not gone, that deserves much study by the medical profession.

I greatly appreciate the honor of being invited to present a paper before this Academy, and it is a source of gratification that a subject which has been fourteen years in developing is at last coming into its own in the eyes of the medical profession.

With a School of Aviation Medicine in the army, with a special course of instruction in it in the navy, with certain medical schools teaching it to their fourth year students, and with 800 civilian and 200 military and naval physicians interested in it, and finally with the comparatively new Aero Medical Association devoted to the furtherance of the subject, I believe it has a great future. Certainly, that future has been enhanced by the recognition you have given it tonight.

PRESENT OPHTHALMOLOGIC STANDARDS FOR COMMERCIAL AVIATION IN THE UNITED STATES.*

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AND

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OUTLINE.

I. Introduction.

II. Discussion of the Present Ocular Standards for Airplane Pilots Set by the Aeronautics Branch of the United States Department of Commerce.

1. Visual Acuity.
2. Depth Perception at Twenty Feet.
3. Ocular Muscle Balance.
4. Inspection of Eyes.
5. Accommodation.
6. Visual Fields.
7. Central Color Vision.
8. Light Sense.
9. Ophthalmoscopic Examination.
10. Psychological Aspects.
11. Visual Reaction Time and Speed of Adjustment.

III. The Effect of Altitude, Flying Stress and High Speed on the Ocular Functions.

IV. Summary and Conclusions.

I. INTRODUCTION.

In 1878[21] Paul Bert published his epoch making studies on the effect of altitude on the human system; in 1896 Langley completed the first heavier-than-air machine. Since these two noteworthy dates the science of aviation has been developed and an International Congress on Aviation Medicine[43] has been held.

*From the Lighthouse Clinic for the Prevention of Blindness, New York, N. Y.

Because the eyes, as soon as the airplane leaves the ground, are subjected to unusual conditions, aviation ophthalmology is one of the most important branches of aviation medicine. Careful judgments must be made at a great distance, and rapid perception is essential because the airplane today travels at a rate of from one to seven miles a minute. A supernormal eye is required in regard to visual acuity, stereopsis, reaction time, perfection of visual fields, accommodation, convergence, divergence, and light and color sense. The eyes must also be resistant to fatigue and able to overcome the changed conditions caused by lack of oxygen. Training is essential because almost everything observed has a changed appearance: shadows are observed from a new angle; tall objects seem short; and the appearance of the terrain is so altered that former visual impressions are of little value without experience. In addition to these factors, the added one of lowered oxygen tension becomes important. At the outset of the World War only an occasional observation of the effect of altitude on ocular functions had been made. As a result of examining six hundred men in the Pierce low pressure chamber and with the Henderson-Pierce rebreathing apparatus,[106] it was found that lowering oxygen tension accentuated any inherent ocular defects. It was also demonstrated that, provided the eyes were normal according to the visual standards set in 1918,[11] it was unnecessary to examine the applicant's eyes during oxygen depletion experiments, because a physical or mental failure would develop before serious eye disturbances or symptoms could be detected.

In this paper it is our purpose to discuss the present visual standards for commercial aviation, to clarify some of the points in the ocular examination, and to compare the requirements for commercial aviation with those for military and naval aviation in the United States. The standards of other countries and the proposed International Standards are also discussed.

II. Discussion of the Present Ocular Standards for Airplane Pilots Set by the Aeronautics Branch of the United States Department of Commerce.

1. visual acuity and refractive errors.

A study of the literature[62 2 11 102 31] shows that the great importance of normal visual acuity for aviators was recognized in an early period by ophthalmologists in all countries. The standards

set by the Department of Commerce of the United States refer to uncorrected vision.

For transport and limited commercial pilots.—The minimum visual requirement for each eye is 20/20. If two or three letters are not read in the 20/20 line, they may be offset by an equal number of letters read in the 20/15 line, and the vision will then be recorded as 20/20.

For industrial pilots.—The minimum visual requirement is 20/30 in each eye; 20/40 may be accepted if the applicant has a correction to 20/20 in his goggles, and provided his depth perception without his goggles is 30 mm. or better.

For private and student pilots.—The minimum visual requirement in each eye is 20/50, provided his depth perception is 30 mm. or better without correction and he wears a correction in his goggles, bringing his vision at least to 20/30.

That these requirements are not too high is confirmed by the statements of many authors[39 28 101] in various countries, but, of course, waivers for vision less than the prescribed standards are commonly granted to experienced pilots who have been able, according to their history, to compensate satisfactorily for their defective visual acuity. Of the 50 per cent of pilots rejected for physical reasons by the Army and Navy, approximately 50 per cent of these are disqualified because of ocular defects.[1]

Apparatus and Methods of Testing Visual Acuity.—The Department of Commerce recommends that at least two Snellen charts should be available, and in order to keep the candidate from memorizing the chart it is well to cut off the bottom, 20/30, 20/20 and 20/15 lines of several charts, numbering them 1, 2, 3, 4, 5. It suggests illumination with one 200-watt or two 100-watt daylight Mazda lamps. During the war, with this arrangement, we found that we obtained at least ten foot-candles of illumination. It would seem to us proper to standardize illumination at ten foot-candles. In our earlier work[11] we found the Ives visual acuity test of value, since this gives not only lines and squares with which to study visual acuity, but a method by which we could easily eliminate malingerers or men who were coached to pass the examination. A candidate who could not see the lines could not indicate their direction with his hand. Special blocks for testing visual acuity have been devised by Hertzog,[66] with which the arrangement of the letters may be quickly changed. (See Fig. 1.)

The Importance of Anisometropia.—The importance of equality of vision is pointed out by Munro,[77] who regards inequality of function between the two eyes as dangerous. Stereoscopic vision

will usually be found better in a man with slightly reduced vision in both eyes than in a man whose vision is defective in one eye only. We have corroborated Howard's[69] findings that inequality of vision often leads to errors of judgment in landing.

The Question of Correcting Lenses. — Whether correction should be ground in the goggles or whether the correction should be incorporated in spectacles worn behind the goggles are important questions. Most ophthalmologists still feel that it is better in principle not to have vision corrected by lenses, at least in aviators in the Army and Navy, as long as it is possible to obtain men with nearly perfect vision. During low-oxygen tests we found that visual acuity diminished in 28 per cent of the normal men examined and in 37.5 per cent of the men who were ocularly

Fig. 1. Letters for testing visual acuity are printed on blocks so that they may be rapidly interchanged. (After Hertzog.)

disqualified for flying. We have found, as Herbolsheimer[65] did, that a man requiring a correction for hyperopia, myopia or even astigmatism, if not of too high degree, may have his vision improve one line if he wears his lenses only part or all of the time.

Goggles.—The following are some of the important points in regard to goggles:[11]

a. *The glass*—

(1) Should have an optically plane surface.

(2) Should have a light transmission of 90 per cent or over for plain white glass.

(3) If tinted lenses are desired, they should be carefully selected according to the conditions which the pilot must meet.

b. *Visual Field.*—It is most important that the aviator have a broad field of vision, and for this reason a large curved glass is desirable. Without a broad field of vision the aviator may not see an airplane appear from the side in time to prevent an accident. Pilots who are doing actual fighting demand a broad visual field above everything else.

c. The goggles should be well ventilated but free from leaks.

Cooper[39] points out (See Fig. 2) the importance of properly centering the lenses of the goggles. He is of the opinion that there are instances when the spectacles may be worn inside the goggles, particularly if the correction is strong. If the correction is weak it should be ground in the goggles whenever possible.

Fig. 2. Arrangement of goggles showing that the pupils are centered behind the nasal third of the goggle lens. (After Cooper.) The interpupillary distance must be fixed by a non-adjustable nose piece so that the optical centers of the corrective lenses can be made to conform permanently with the primary visual axes.

The Effect of Altitude on Visual Acuity.—The most important ocular symptom produced by altitude is failing vision. The pilot often complains that he cannot see the ground clearly in landing and that he has difficulty in picking out enemy planes. In 1918[11] we studied visual acuity by the use of the Ives test object and Johnson's visual acuity test apparatus, as well as by the usual Snellen test type.

Forty-four subjects were examined with the rebreathing apparatus and in the low pressure chamber. They were classified

as normal or subnormal—i. e., those who could pass the examination for the Department of Military Aeronautics and those who would be ocularly disqualified. The thirteen subnormal subjects were so classified because of defective vision arising from errors of refraction.

The Effect of Tobacco on Visual Acuity.—The effect of tobacco[11] upon the visual acuity has also been studied. The effect of smoking one strong cigar or inhaling one or two cigarettes was compared with control tests. Seventy-five per cent of the men tested showed decrease in visual acuity, 6 per cent showed a rise, and 19 per cent showed no change. We found at the Medical Research Laboratory, Issoudun, France, that inhaling two cigarettes often affects the visual acuity more markedly than drinking five ounces of cognac brandy.

Discussion.—Normal visual acuity is most important for all aviators, especially until they are trained by practice to see accurately when flying. Exceptional visual acuity is a valuable asset for the military aviator. The standards set by the Department of Commerce, although they possibly exclude men who would make safe pilots, should not be lowered.

2. DEPTH PERCEPTION AT TWENTY FEET.

The prime importance of rapid and accurate judgment of distance is conceded by all authorities, but the method by which the aviator judges distance and the best method of testing his ability to judge distance has been a subject of controversy. Although there is no doubt that accurate estimation of the length and breadth of an object can be formed by one eye, all authorities agree that depth and solidity of an object is more accurately estimated with two eyes.

The binocular parallactic angle is important in this latter estimation, but most authorities now agree with Halben,[62] one of the earliest students of this problem, that binocular parallactic displacement plays little part at the great distance at which judgments must be made in flying.

Other things being equal, we believe that the man who has true stereoscopic vision and is able to make his estimations rapidly is the safer man in flying and will be more efficient in combat work. It may be significant that special examinations made of seventy monitors, eight testers, one hundred and twelve fliers from the American front, twelve French fliers and twenty-one American aces, revealed that all the men examined had normal stereoscopic

vision, and most of them made their judgments quickly and accurately. In England, Brailey[27] believes that the parallactic sense is of greatest importance in making successful landings and the stereoscopic sense is of little value. In discussing this paper, Clements did not entirely agree with Brailey and stated that men who had been flying, who had only one eye, had learned quasi-stereoscopic vision long before.

Standards for Stereoscopic Vision for Commercial Aviators.—The United States Department of Commerce requires a depth perception of not more than 30 mm., without correcting glasses, as the average of all trials for the five classes of pilots. If the candidate wears correcting lenses, the depth perception will be tested with his glasses in addition, and in such a case the candidate will not be qualified unless his depth perception is 30 mm. or less both with and without correction. When the trials are nearly approximate in result, three trials are considered sufficient, but if they are widely variant, not less than ten trials should be given and the ten averaged.

Some authorities are of the opinion that this standard is too high, but Deyo,[45] who studied a group of qualified and disqualified pilots, found, using a six meter stereoscope, that men who were ocularly qualified averaged 11.93 mm. From this she concludes that it is probable that the standards should be raised, and a minimum depth perception average of 25 or 20 mm. should be required.

As an international standard, Onfray[82] suggests that the candidate should read without error stereoscopic charts in which the parallax is not greater than 30 or 40 seconds. With an apparatus with three points, similar to the Howard-Dolman apparatus, the error should not be greater than 50 millimeters.

Visual Acuity and Anisometropia in the Judgment of Distance.—Visual acuity is an important factor in stereoscopic vision. Deyo[45] tested a group of men with the Howard-Dolman apparatus. Fifty-seven men with 20/15 vision showed an average depth perception of 12.02 mm., and sixteen men with 20/20 vision in both eyes showed a depth perception of 28.11. These findings may indicate that visual acuity is an important factor in depth perception.

That inequality in function between the two eyes is more dangerous than lowered visual acuity in both eyes is pointed out by Munro,[77] and we have long believed that, all things being equal, the man with 20/30 vision in each eye was a safer pilot than one with 20/30 in one eye and 20/20 in the other.

The Question of Monocular Vision and Judgment of Distance.—Although Howard[68] and Deyo[45] have done valuable experimental work which shows the great importance of both eyes in true stereoscopic vision, Halben[62] and Doyne[50] both believe that a one-eyed man, after he has learned to judge distances with one eye alone, is less dangerous for flying than a two-eyed man whose eyes may be unreliable.

Judgment of Distance in Landing.—Authorities seem to agree that the most difficult test in the judgment of distance is in landing, especially landing on water. When the water is smooth the aviator has little by which to judge his distance except the motion parallax of his plane with the horizon, and if there should be a fog even this aid is wanting. This would seem to indicate that we are aided in judging distance in landing by objects on the ground not only from the parallax which they give but also the relative size of the shadows they cast. Aviators who land high and pancake habitually place the movable object in the two or three point stereoscope in front of the other object or objects, whereas the men who strike the ground at an angle usually place the movable object posterior to the fixed object or objects.

Defective Stereopsis and Training.—It is pointed out by Davis[42] that defective stereoscopic vision may result from an attack of influenza or any prolonged close application of the eyes. Applicants who have poor stereoscopic vision can sometimes be greatly benefited by exercises and at times their depth perception so improved that they can pass a test.

As an example of what orthoptic training will do, the following record is appended:

W. G., aged 33 years, an ace in the German Air Service in 1916, was examined for a transport pilot license in December, 1927. His vision without correction was 20/30 in the right eye and 20/20 +5 in the left eye. Measured with the Howard-Dolman apparatus his depth perception was 56 mm. (average reading). Referring him for orthoptic training, we received the following report from Dr. John Dunnington:

"December 17, 1927. W. G. was recently rejected by Dr. Berens for transport air license on account of vision of 20/30 and a defective depth perception. Has been flying since 1915 and has had no real crashes. Vision in the right eye 20/30, left eye 20/20, at 6 meters: left hyperphoria 1$^{\Delta}$; exophoria 1$^{\Delta}$; prism divergence 6$^{\Delta}$; prism convergence 10$^{\Delta}$. At 25 centimeters: exophoria 15$^{\Delta}$;

left hyperphoria 1$^{\Delta}$; near point of convergence to the intercentral base line 100 millimeters. Imperfect fusion with stereoscope.

"December 19, 1927. To start with E. series in Wells' charts.

"December 23, 1927. Continue exercises. Fusion somewhat improved.

"December 30, 1927. To supplement fusion exercises with approximation 15 times twice a day. Report in ten days.

"January 9, 1928.

Accepts right eye + .50 $\bigcirc$ +1 axis 105° V. equals 20/20—3
left eye +.50 axis 75° V. equals 20/20

At six meters: left hyperphoria ½$^{\Delta}$; exophoria 1$^{\Delta}$; prism divergence 8$^{\Delta}$; prism convergence 15$^{\Delta}$; at 25 centimeters: exophoria 10$^{\Delta}$; near point of convergence to the intercentral base line, 95 mm.

"January 10, 1928. Passed the examination. To use stereoscopic exercises when necessary."

Returning on January 10, 1928, his depth perception, measured with the Howard-Dolman apparatus, was 19 mm. (average reading). Prism divergence, 5$^{\Delta}$; prism convergence, 16$^{\Delta}$.

This pilot was re-examined every six months and the depth perception varied from 15 mm. to 24 mm. until January, 1931. At this time the depth perception was 54 mm.; prism convergence, 15$^{\Delta}$; prism divergence, 13$^{\Delta}$. He was advised to work with the stereoscope once more. In July, 1931, his depth perception was 21 mm. He said that he had been working with the stereoscope just previous to taking the test.

The Effect of Altitude on Depth Perception.—It is stated by Howard[68] that "the effect of low oxygen tension on the depth perceiving sense is of little importance in aviation because the critical time during the flight is in landing, when there is no lack of oxygen." He apparently does not consider the fact that a man who has flown at a high altitude for any length of time may suddenly be forced to land with a dead stick and have no time for his ocular functions to readjust. One of us (C. B.) has known of several crashes which were undoubtedly due to inability of the flier to judge distance properly because of just such a situation. The fact that aviators know that it takes time for their eyes to readjust, in judging distance, is one of the reasons why experienced pilots always insist upon making several turns of the field before landing.

The Value of Orthoptic Training on Ability to Judge Distance.—During the war, exercises were given at the School of Aviation Medicine at Mitchell Field and in the Research Laboratory, Issoudun, France, to those men who had difficulty in judging distance or made bad landings. Emphasis was placed upon improving prism diverging and converging power by the use of prisms and a stereoscope. A modified Howe ergograph was used for giving diverging and converging exercises with prisms and later, without prisms, and also for exercising accommodation.

In England, during the war, Wing Commander Clements directed the center, which was noted for helping men who had muscle imbalance and difficulty in landing. It is significant that Deyo[45] found, in the group disqualified for flying because of muscle imbalance, color blindness, low angle of convergence and poor visual acuity, that the average depth perception was 36.30. In the study of a qualified group the average was 11.93.

Apparatus and Methods of Testing Depth Perception.—It was soon appreciated by all countries engaged in the World War that the ordinary stereoscope was not a satisfactory method for studying stereoscopic vision, although it acted as a means of determining whether a man did or did not have stereoscopic vision. The test ordinarily employed was not sufficiently accurate, and it did not lend itself readily to the task of accurately classifying subjects in regard to their relative ability. The apparatus advised by the United States Department of Commerce is the Howard-Dolman modification of the Brooks Bank James apparatus, 1908, England.[68]

The Howard-Dolman depth perception apparatus is placed against the wall at a distance of twenty feet. It must have good overhead illumination; care must be taken to have the zero line of both rods equidistant from the applicant. The box should be placed at such a height that the applicant can see only the center of the rods through the window in the front of the apparatus.

Procedure.—The rods in the box are widely separated by the examiner and the examinee is required to manipulate the two cords until he brings the movable rod into juxtaposition with the fixed one making both appear the same distance from him. The test is repeated several times, the rods being widely separated before each trial. The examinee's estimation of depth difference is read in millimeters directly from the scale and entered on the record. The candidate will be given only twenty seconds to make the adjustment at each trial.

Precautions.—No information concerning the results of the successive trials will be given the examinee until after completion of the test. He is required to hold his head straight and not to one side or the other. The

examinee will not be allowed to seesaw the rods back and forth the full depth of the box for this gives information as to the length of the box. The rods should be adjusted promptly since the time element is an important factor. Care will be taken by the examiner:

To avoid casting a shadow on the background.

To avoid so placing the hands as to give the examinee information as to his error.

To avoid any facial expression from which the examinee might gain information as to the result of his efforts.

When the examiner is separating the rods the cord should be removed from the hands of the candidate, so that he cannot judge the distance the rods have been moved by the length of cord that has slipped through his fingers.

The United States Army and Navy now require the use of the Howard-Dolman apparatus. The stereomicrometer is accurate for testing stereopsis and does not require so much space.[69a]

We believe that it is important to consider the time factor in making judgments of distance and have devised a six-meter recording stereoscope. (See Fig. 3.) A push button *(A)*, attached to a six-meter cord *(B)*, permits the instrument to be used at any desired distance. Milk-glass plates *(C)* are used instead of rods. Records are made with a continuous roll of paper *(D)*. The records of one man who had excellent stereoscopic ability may be seen by referring to Fig. 4. The single horizontal line indicates the position of the stationary test object, and it shows how accurate this man was in his judgments.

Apparatus and Method Suggested for International Standards.—Onfray[82] suggests the use of the diploscope, the length of the apparatus not being stated; also, the use of a stereoscope and stereoscopic charts, in addition to the apparatus with three points, similar to the Howard-Dolman test. Cantonnet[31] also advises the use of a six-meter stereoscope.

Discussion.—The present requirement for stereopsis set by the Department of Commerce should be maintained because men who habitually use both eyes and who have normal eyes can usually average well within thirty millimeters. If the required average cannot be attained, the cause should be determined and eliminated or overcome by orthoptic training before the applicant is accepted. There is no doubt that a trained one-eyed man can land a plane—which is accomplished more by the motion parallax than the binocular parallactic angle—and is usually safer than the man who normally uses both eyes but has defects which may impair coordination at the important time of landing. Onfray[82]

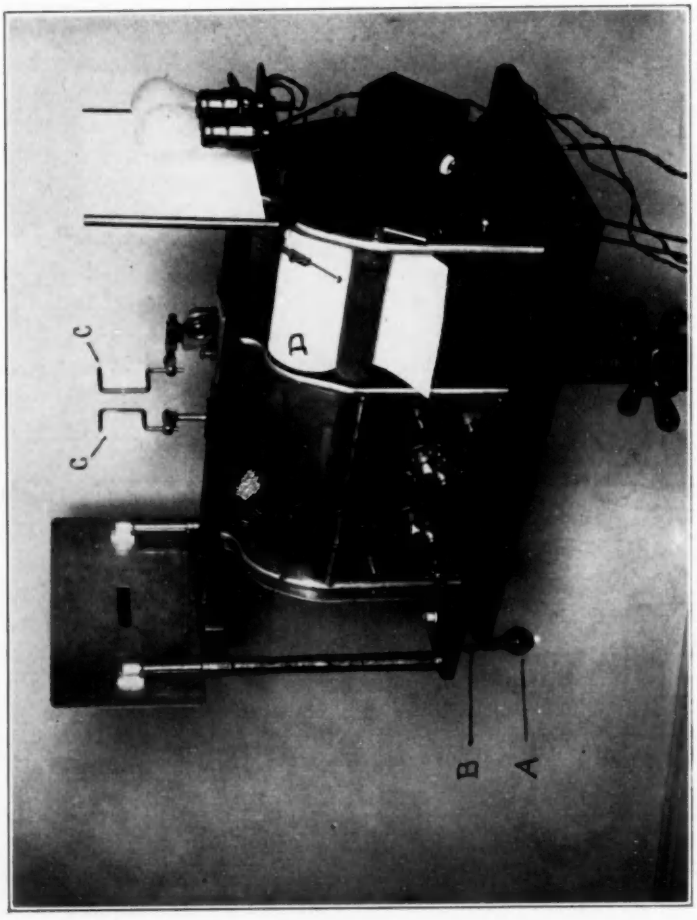

Fig. 3. A six meter recording stereoscope which automatically controls the time factor. A, Push button. B, Six meter cord. C, Milk-glass plates as test objects. D, Continuous roll of paper for recording judgments.

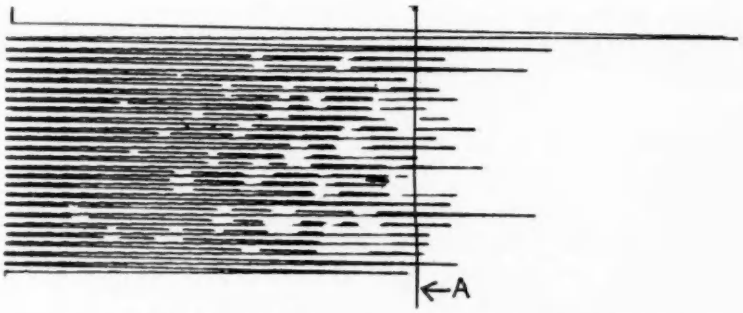

Fig. 4. Record of stereopsis of a man who has had excellent training in studying aerial maps with a stereoscope. Line A indicates the position of the stationary test object. Record made with the six meter stereoscope (see Fig. 3).

proposes 50 mm. as an international standard for the allowable error, and certainly experienced judgment should be obtained before too hastily excluding a man because of faulty stereopsis.

3. OCULAR MUSCLE BALANCE.

The Importance of Normal Muscle Balance.—In this country the importance of orthophoria is still insisted upon by Davis,[42] although Stitt[93] points out that the English Flight Surgeons do not stress eye muscle balance as do the specialists in our Army and Navy. In spite of the fact that the permissible 0.5$^{\Delta}$ of hyperphoria has been increased to 0.75$^{\Delta}$ in our Navy, out of 2,404 men examined in 1927 at Pensacola, fifty-four (2.2 per cent) were disqualified on account of this difficulty. Davis points to the variability of hyperphoria, and remarks that the study of fatal accidents indicates that slight degrees of hyperphoria seem to have had no bearing. He also states that the specialists at Pensacola have failed to note any superiority in student pilots who barely passed the various eye tests when compared to those who have few or no defects.

Although we should not underestimate the importance of poor coordination and slow reaction time, I still believe that the ability of the eyes to function normally under the stress of flying and low oxygen tension is an important asset, especially to a man who is accustomed to using both eyes. Also, I believe that latent heterophoria often becomes manifest or results in diplopia under flying conditions.

Apparatus, Tests and Methods Used by the Department of Commerce.—These consist of—

A spot light or candle; a trial frame; a plane red glass for the trial frame; prisms as follows: 0.5, 1, 2, 3, 4, 5, 6, 8, 10, and 12 prism diopters. (Strengths between these or above 12 may be obtained by combining two prisms. For example, if 14 diopters is desired, the 12 and 2 diopter prisms may be combined); a Maddox multiple red rod.

Test for diplopia—Procedure.—The applicant is seated twenty feet distant and facing the candle or spot light. The trial frame will be adjusted and a plane red glass placed in front of the right eye. The spot light or candle will then be lighted. The applicant looking straight ahead (both eyes open) will be asked whether he sees one light or two; if two, the relation of the red light to the white one. His head is then lowered so that in order to see the light he must look through the top of the red glass and trial frame (he must not by any chance be able to look over the top of the red glass). The same questions are repeated. The head is then raised so that he looks through the bottom of the glass; then to the left so that he looks through the right; then up and to the left; down and to the left; up and to the right; down and to the right, so that he looks successively through the diagonally opposite sides of the glass.

If at any time he sees two lights, he has diplopia. With the red glass

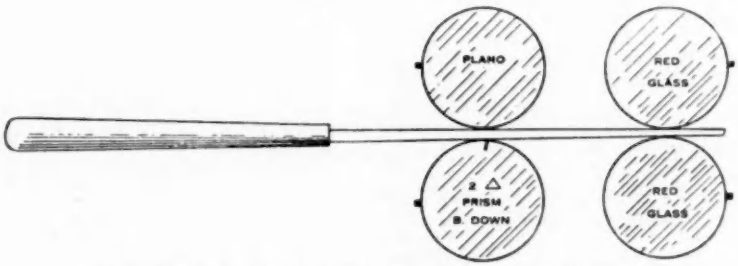

Fig. 5. A new instrument for testing for diplopia. (After Herbolsheimer.)

in front of the right eye, if the red light is to the right of the white one, the diplopia is homonymous; if to the left, it is crossed. As a check on the veracity of the candidate, a 4△ prism should be placed occasionally base up or base down in front of the red glass. This will produce a diplopia. Great care should be taken in selecting the red glass to be sure it is optically plane or diplopia may be produced.

A new instrument for testing for diplopia has been devised by Herbolsheimer.[65] (See Fig. 5.)

Hyperphoria.—For transport and limited commercial pilots. Procedure.—The applicant is seated twenty feet distant from the spot light or candle, the trial frame is adjusted and a Maddox rod (which should have been tested) placed in front of the right eye. The left eye is covered and the applicant instructed to adjust the rod until the line of light is horizontal. The left eye is then uncovered and he is asked to locate the line in reference to the light. If the line passes through the light, the findings will be recorded as zero. If the line is below the light, he has right hy-

perphoria. If it is above, he has left hyperphoria. A 0.5$^{\Delta}$ prism will then be placed before the Maddox rod (held in the fingers) base down if the line is below the light, base up, if above, and the applicant again asked to locate the line in reference to the light. If it is still below, a 1$^{\Delta}$ prism is placed before the eye replacing the 0.5$^{\Delta}$ prism. This procedure is continued with prisms increasing in strength until the line passes through the light. If one prism fails to correct, and the next overcorrects—that is, the line passes to the opposite side of the light—the reading is between the strength of the two prisms. For example: 0.5$^{\Delta}$ fails to correct, 1$^{\Delta}$ overcorrects. The reading will be made as 0.75$^{\Delta}$.

Precaution.—When the candidate is suspected of not being frank in his answers, the test may be done by placing a 1$^{\Delta}$ prism base up or base down in front of the Maddox rod. This will produce a hyperphoria, and the line should appear above or below the light. The strength of the prism may be gradually reduced to zero, when the line should pass through the light if the candidate has no hyperphoria.

Vergence tests—For transport and limited commercial pilots—Procedure.—The applicant will be seated twenty feet from the spot light or candle. Prism divergence will be tested first. A prism of 1$^{\Delta}$ will be placed before the right eye, base in, and the applicant asked whether he sees one light or two. Prisms of increasing strength are then placed before the eye until the applicant is no longer able to fuse the light and he sees two. Prism divergence is measured by the strength of the last prism that he is able to overcome. Prism convergence is then tested in the same way, but by placing the prisms base out and starting with a strength of 2$^{\Delta}$. Prism convergence will be measured by the strength of the last prism the applicant is able to overcome.

Standards set by the Department of Commerce.[98]—Men who develop diplopia with the red glass test are disqualified for any class of flying. They are not disqualified if diplopia develops only at the extreme limits of the field.

No other test of ocular muscle balance is required of industrial, private, and student pilots, unless a diplopia develops in other than the extreme positions, in which case the tests for hyperphoria and prism vergence will be performed. In such cases the results of the examination are forwarded to the medical director and no certificate or permit is issued.

An applicant with the red glass before the right eye should be able to maintain a binocular single vision of forty-five degrees in all directions from the central position. Diplopia occurring beyond forty-five degrees in any direction from the central position will not be considered disqualifying.

Tests for hyperphoria and prism vergence are required for transport and limited commercial pilots irrespective of the presence or absence of diplopia.

Interpretation of findings.—Hyperphoria of more than 1 diopter will disqualify a man as transport or limited commercial pilot. Prism convergence of eight diopters or more is satisfactory provided prism divergence is not less than four diopters. Prism divergence of four diopters is re-

quired but more than four diopters may be accepted, provided prism convergence is increased in proportion.

Disqualifying Defects in Muscle Balance and Their Treatment.—Campbell[30] believes that heterophoria is a common cause of eye strain and momentary diplopia in the lateral and, at times, in the primary positions. Uncertainty of binocular vision, inaccuracy in judging distance, lessened endurance, a feeling of tenseness and discomfort about the eyes have all been attributed to imbalance of the ocular muscles. Ocular muscle imbalance occurring among applicants for flying training has been studied by Thorne.[96] In his review of the examinations of 500 men, only those cases exhibiting two or more diopters of esophoria, one or more of exophoria and one-half or more of hyperphoria are considered.

	No.	%
Total number of aviators examined by Thorne	500	1.
Horizontal imbalance:		
Esophoria	152	62.29
Exophoria	92	37.7
Total	244	48.8
Vertical imbalance:		
Right hyperphoria	39	7.
Left hyperphoria	92	37.7
Total	132	25.6

Of these 500 applicants examined, 348, or 69.7 per cent, were relieved from flying training because of inability to make proper progress.

Of the 244 exhibiting a horizontal imbalance, 127, or 72.5 per cent, were eliminated. Of the esophorias alone 103, or 67.7 per cent; and of the exophorias alone, 74, or 80.4 per cent failed. He concludes: "Whether or not the ocular muscle imbalance played a part in the elimination of these students is open to discussion."

In studying the effect of low-oxygen tension produced artificially, 50 per cent of the men who could not pass the aviation examination of the army showed a decrease in the field of fixation, which was most marked above, whereas only 7.37 per cent of those who could pass the examination showed a limitation in the field of binocular fixation. The entire question of disqualifying eye muscle imbalances in aviators has been studied by Loy.[74]

The association between prism divergence, the esophorias and exophorias, is easily visualized by Loy's diagram. (See Fig. 6.)

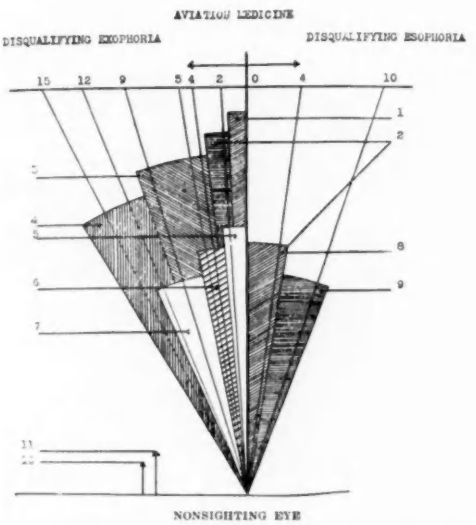

Fig. 6. Diagram of disqualifying heterophoria. (After Loy.)

1. Prism divergence less than 2 D.
2. Prism divergence less than 4 D, associated with esophoria greater than 4 D.
3. Prism divergence greater than 9 D, associated with angle of convergence near 40.
4. Prism divergence greater than 15 D.
5. Exophoria greater than 2 D at 6 meters, associated with (1) angle of convergence near 40, or (2) diplopia in the lateral positions on the tangent curtain.
6. Exophoria greater than 5 D at 6 meters.
7. Exophoria greater than 12 D at 33 centimeters.
8. Exophoria greater than 4 D at 6 meters, associated with (1) diplopia in the lateral positions on the tangent curtain, or (2) accommodation 2 D below or above that given for normal for age in Duane chart, or (3) hyperopia near the disqualifying limit, or (4) prism divergence less than 4 D.
9. Esophoria greater than 10 D at 6 meters.
10. Hyperphoria greater than 0.75 D is disqualifying for applicants.
11. Hyperphoria of 1 D is allowed for qualified aviators.

Note—Normal exophoria at 33 centimeters is 4 D: normal exophoria at 6 meters is 0: normal esophoria at 6 meters is 0, but unassociated with other conditions, is not disqualifying below 10 D.

The Treatment of Disqualifying Defects.—Experience in this country and in France[15] made us believe that orthoptic training is of value in heterophoria, and that the results are sometimes permanent has also been pointed out.[19]

During the war, by the methods used by Clements,[33] 80 per cent of successes were recorded as a result of eye training, and since the war, by treating only suitable cases this percentage has been increased. In this country Cooper[39] points out that the Depart-

ment of Commerce refers all men with ocular muscle imbalance, and even those with moderately poor vision, to competent ophthalmologists for treatment. Many of these men exhibit normal eye muscle balance several weeks or months after the proper treatment of these defects.

That muscle balance may be taken too seriously in certain cases is possibly illustrated by the following case report:

H. S. S., Jr., aged 26 years, wanted to enlist in the Army Air Service and was told that his depth perception was deficient. The report from the Army official was as follows:

"December 16, 1929. Vision, right eye 20/30+8, left eye 20/30 +7. Depth perception 101 millimeters (average reading). At 6 meters: exophoria, 3$^{\Delta}$; left hyperphoria, 1.5$^{\Delta}$; at 25 centimeters; exophoria 24$^{\Delta}$."

Our findings on January 2, 1930, showed that his depth perception was 43 millimeters, as measured by the Howard-Dolman apparatus. Vision, right eye was 20/20+, left eye 20/15—, exophoria, 1½$^{\Delta}$; at 6 meters: left hyperphoria, 0$^{\Delta}$; at 25 centimeters: exophoria, 8$^{\Delta}$; prism divergence, 5$^{\Delta}$. Under a cycloplegic he accepted no lens in either eye. He was advised to work with a stereoscope and Wells charts, as well as to exercise with prisms.

April 15, 1930. Vision, right eye 20/30, left eye 20/30+8. Depth perception, 17.8 millimeters; exophoria, 1$^{\Delta}$; at 6 meters: left hyperphoria, 75$^{\Delta}$; at 25 centimeters: exophoria, 3$^{\Delta}$; prism divergence, 22$^{\Delta}$.

August 31, 1931, he returned for re-examination. Vision in his right eye was 20/20+ and in left eye 20/15—. His near points of accommodation were as follows: He read the 300 millimeter print at 120 millimeters in each eye. His depth perception was 24 millimeters. With the Maddox rod he had exophoria, 1$^{\Delta}$; 25 centimeters, exophoria, 10$^{\Delta}$. Testing by screen and parallax he had orthophoria at six meters and exophoria of 15$^{\Delta}$ at 25 centimeters. There was possibly a left hyperphoria of ½$^{\Delta}$ at six meters. He is now considered by us to be a good flying risk, as far as his eyes are concerned, but the Army requirements demand an exophoria at the near point not over 10$^{\Delta}$ and at his last Army examination he had 12$^{\Delta}$.

Prism Vergence and the Near Point of Convergence.—It seems unfortunate that the Department of Commerce does not require study of the near point of convergence, the importance and utility of which is still maintained by Lawrence[71] and a test which is still

used by the British.[28] It would seem wise, as one of us[14] has pointed out, to consider a near point of convergence of over 80 millimeters as a cause of disqualification, at least until the near point can be brought within the normal limit or the cause of the insufficiency of convergence detected. The highest average figure[15] that we found in the study of 224 aviators was 66 millimeters, and this corresponded with an age average of 31 years.

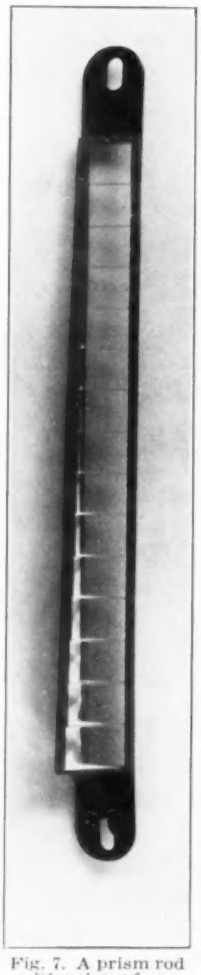

Fig. 7. A prism rod with prisms from 1Δ to 45Δ.

The study of prism convergence and divergence at six meters and twenty-five centimeters is certainly of value, and this examination is facilitated by using prisms in a rod. A prism rod* has been constructed in which the usual size of the prism has been reduced, making a short rod easily handled, even though the strongest prism is 45Δ. (See Fig. 7.) Work has been done by Buchanan[29] in the comparison of two methods of applying prism tests to the eyes.

The Effect of Altitude on the Near Point of Convergence.—If the near point of convergence falls off markedly during flying, the aviator's ability to make landings properly will be impaired, and, therefore, the near point of convergence has been taken during the rebreathing test and low-pressure chamber experiments.

One hundred and forty-seven men with normal eyes were examined on the rebreathing apparatus according to the requirements of the Air Service of the United States in 1918.

Decrease in converging power, 50.3 per cent.

Increase in converging power, 17.6 per cent.

Fluctuation in converging power, 11.5 per cent.

No change in converging power, 20.6 per cent.

*The rod may be purchased from A. Haustetter, 2 East 45th Street, New York.

Of eleven subnormal men examined, six were disqualified for visual acuity and five for muscular imbalance; 45.7 per cent showed decrease in power of convergence. Increased converging power, fluctuating changes and no change in the near point of convergence were each noted in 18.1 per cent. Of sixteen normal men examined in the low-pressure chamber, 50 per cent remained unchanged. In the subnormal group the recession of the near point of convergence was very marked, sometimes resulting in diplopia.

The results indicate that the rebreathing apparatus and low-pressure chamber give almost identical findings, and in each case the determining factor seems to be the lowering of oxygen tension as the administration of oxygen soon causes the convergence near point to return to normal, irrespective of the barometric pressure.

Fatigue of convergence has been studied with Howe's ophthalmic ergograph. The findings on the rebreathing apparatus and in the low-pressure chamber showed a more rapid onset of fatigue than occurred with the controls. At 15,000 feet and at 20,000 feet the fatigue was marked. Here also the administration of oxygen caused a rapid return of converging power.

We have also shown that the fatigue which is developed in normal subjects under the condition of altitude artificially produced occurs earlier in men who are subnormal, according to the ocular standards set by the United States Army.

The Effect of Altitude on Muscle Balance and Muscle Strength.—To determine the effect of lack of oxygen upon the ocular muscles thirty-five men, acceptable for the Air Service, have been examined on the rebreathing apparatus and the findings checked by repeating the test in the low-pressure chamber. Prism vergence was taken at sea level, 5,000, 10,000, 15,000 and 20,000 feet, and at this point oxygen was given in the low-pressure chamber for five minutes, and on the rebreathing apparatus the mouthpiece was taken out, allowing the subject to breathe normally.

In all the subnormal subjects examined, particularly those with convergence insufficiency alone or combined with divergence excess, there was a marked loss in the power of prism convergence, and diplopia often occurred between 10,000 and 15,000 feet. Men with more than one degree of hyperphoria, particularly when combined with exophoria, showed a rapid reduction in muscle strength, often resulting in diplopia.

Discussion of the Present Standards Set by the Department of Commerce.—The Department of Commerce has been wise in not insisting too strongly on perfect muscle balance, relying more on performance tests. Some of the difficulties inherent in the use of the Maddox rod have been pointed out by Dolman,[47 49] and the combination of the Maddox rod screen test,[47] suggested by Dolman, removed some of the objections to the use of the Maddox rod.

In the international standards suggested by Onfray,[82] the use of the Remy diploscope is advised and normal muscle balance is required. For examiners who have had experience, the screen and parallax test is sufficient without the use of the Maddox rod; but this should be used both at six meters and twenty-five centimeters and not only at the near point, as suggested by the British Air Ministry.[28] The present tests required by the Department of Commerce will not always exclude men with divergence excess, and it is possible that the screen and parallax test, used at six meters and twenty-five centimeters, should be added for all classes of pilots. It is unnecessary to measure all cases with prisms, and, if marked tendency to esophoria and exophoria is noted, the actual deviation should be recorded and these results compared with ability to judge distance and all other ocular conditions in determining the candidate's fitness to fly.

Defects in the present regulations are recognized by Cooper,[39] who states that in using the red-glass test for diplopia one should determine the veracity of the applicant's statement, when the slightest doubt arises, by testing for hyperphoria, determining prism convergence and divergence, and comparing these values with the normal. He also recommends the use of the cover test and careful examination for deviation. He suggests placing a red glass with a 0.5^{Δ} prism, base up or down, before the eye. He states that this should produce vertical diplopia and that the answers given by the applicants can be verified by the known position of the prism. Some men may be able to fuse with a 0.5^{Δ} prism even though a red glass is used, especially if they have used prism exercises.

4. INSPECTION OF THE EYES.

Whenever it is possible the eyes are inspected by bright daylight. The following conditions must not be overlooked:

Eyelids: Ptosis, blepharitis, trichiasis, entropion, ectropion and chalazion. Lagophthalmos from slight facial paralysis or injury should also be looked for.

Muscles: Considered under Muscle Balance.

Lacrimal apparatus: Imperfect drainage due to stenosis or improper contact of the puncta with the bulbar conjunctiva. Excessive lacrimation and chronic dacryocystitis should also be noted.

Conjunctiva: Trachoma and old scars. Chronic conjunctivitis should be cured before the applicant is accepted.

Cornea: Opacities, pannus and pterygium.

Pupils: The examination should be conducted in a darkened room. Unequal size, irregular shape, and failure to react to light and accommodation should be carefully noted.

The importance of studying the iris reactions is emphasized by Cantonnet.[31] Because of the danger of admitting a man with tabes or general paralysis, he believes that posterior synechiæ should be looked for, as these are often caused by syphilitic iritis.

According to the Department of Commerce[98] any pathological condition which may become worse or interfere with the proper functioning of the eyes under the fatigue and exposure of flying disqualifies; nystagmus or strabismus also disqualifies.

A candidate possessing any of the defects mentioned above should be disqualified if the condition cannot be remedied, since these defects often prove to be a serious detriment while flying.

5. ACCOMMODATION.

Importance of Normal Accommodation.—The importance of accommodation and the weakening effect of altitude on accommodation was one of the earliest ocular symptoms complained of by balloonists.

Normal accommodation is of value, as tested by the usual method, by fatigue tests and as it is affected by altitude because of the constant and rapid changes in accommodation necessitated in flying. Rapid adjustment is required when the aviator looks from his instruments and maps after observing other airplanes and other distant objects. Failure of the pilot to accommodate readily may make it impossible for him to read his instruments, or the sensations produced in attempting to read them may cause him to neglect their study.

Standards for Accommodation Set by the United States Department of Commerce.—A fair degree of accommodation is required for transport and limited commercial pilots only and correction in the goggles may be worn while the test is being made, provided the vision is below 20/20 in

either eye. The requirements for transport and limited commercial pilots are that they have at least two diopters of accommodation, that is, that they can read Jaeger No. 1 or 2 test type at a distance of not more than thirteen inches.

Speed of Accommodation.—The tachistoscope devised by Ferree[53] has been used by Goodall[59] to study accommodation. He concludes that accommodation is of importance in the rapid change in focus required in landing a plane and in pursuit work.

The Effect of Altitude on Accommodation.—We found that fatigue, especially that produced by the habit of late hours,[11] was probably the most important factor in causing early failure in accommodation in men who normally showed no accommodative weakness when altitude was produced artificially.

We began our scientific investigation of this problem in 1918.[11]

Approximately 45 per cent of the normal group (qualified for the Army Air Service) showed decrease in the power of accommodation in both methods of testing. In the subnormal group (disqualified for the Army Air Service) 64 per cent responded by recession of the near point in the rebreathing test, and the three men examined in the low-pressure chamber exhibited failure of accommodation.

From these studies it is believed that aviators who have less than 2 diopters of hyperopia and less than 0.5 diopter of hyperopic astigmatism will not show marked changes in accommodation before their efficiency is seriously impaired by circulatory or psychological disturbances due to lack of oxygen.

Fatigue of accommodation as affected by low oxygen tension was also studied with Howe's ophthalmic ergograph and the first three modifications of his instrument. Three-minute tests with three-minute intervals were made without the low oxygen tension effect; then three-minute runs with the same time intervals were made in the low-pressure chamber and with the rebreathing apparatus. The findings with the rebreathing apparatus and with the low-pressure chamber showed, at 15,000 feet, a more rapid onset of fatigue than was evidenced by controls, and at 20,000 feet fatigue was evident by recession of the near point.

Discussion of the Present Standards Set by the Department of Commerce.—Onfray[82] does not consider the matter of accommodation an essential factor as an international standard, except as it pertains to other tests.

The United States Navy requires normal accommodation, according to a special age table, and in the United States Army the

applicant must read Jaeger No. 1 type to qualify for class I and Jaeger 1.5 type for classes II and III. This test may be made with correction in goggles, provided the man is a rated pilot or observer.

Test objects vary greatly in results obtained and we[13] have studied the comparative value of each.

The Duane disk is the best test object for general use. It is found, however, that during the rebreathing test it is difficult for the subject to recognize quickly the faint black line on the disk, and the later readings are not satisfactory.

Fig. 8. A new accommodation chart.

The near point of accommodation should be taken with each eye separately, using 400 millimeter type and an illiterate "E" in testing the near point. An accurate accommodation chart (see Fig. 8) has been drawn and photographically reduced. This chart includes capital letters only.

The results of our[20] tests for fatigue of accommodation do not justify accepting the Duane standards for accommodation as a final criterion of ability to accommodate without rapid fatigue.

The Duane[51] standard for all ages should, however, be required for all aviators by testing the ability to read 400 millimeter print, while wearing their correction, if correction is permitted by the Department of Commerce.

If, after testing by the Duane standard, disqualifying defects are found, in each case the cause should be ascertained and the accommodation rectified until by the same standard it becomes normal. No man should be allowed a pilot's license until this is done, as failure of accommodation may endanger lives of others. The elimination of the cause of the defect and special exercises to improve accommodation will be found of benefit in many cases.

6. VISUAL FIELDS.

The widest possible range of peripheral vision is necessary for safety in aviation. The aviator[39] must be able to detect objects within his peripheral vision while looking straight ahead. Fortunately, the periphery of the retina is very sensitive to moving objects. The construction of goggles and the airplane should restrict the field as little as possible.

Standards for Visual Fields Set by the Department of Commerce Compared with the Army, Navy, and International Requirements.—The confrontation test with the fingers as test objects is used in the examination of all classes of pilots after the first examination, but perimetric records are required if lesions are found or suspected and perimetric charts are required for transport and limited commercial pilots.

The meridians studied in each eye for form and color are 0°, 45°, 90°, 135°, 180°, 225°, 270° and 315°. The color fields for red and blue with a 5 mm. test object of standard color should be nearly concentric with the form field. The normal field for a 5 mm. test object extends on the temporal side to about 90°, or more; upward, 55°; inward, 60°; downward, 70°. Any contraction of the color field or of the form field, and other pathological alterations (nasal contraction, scotomata, etc.) are disqualifying for all classes, but industrial, private and student pilots will not be disqualified if the contraction or scotomata are not marked.

The same standards obtain in the Army and Navy. The international standards proposed by Onfray[82] call for normal visual fields, and the United States Army and Navy require normal visual fields as tested by a perimeter.

Methods of Testing.—Except in instances mentioned above, the United States Department of Commerce[98] requires the use of a perimeter in testing visual fields. A stereocampimeter may be used instead of the perimeter only if it records the complete visual field. Each eye is tested separately. The examinee's head is supported on a chin rest, one eye covered, and the other fixed upon the white spot located in the center of the arc of the perimeter. The test object first used is a 5 mm. white circle,

carried along the inner surface of the perimeter arc, from without inward. The points where the test object is first seen, in the different principal meridians, are marked upon diagrams of the normal field.

The white test object gives the form of the visual field. The two colors —red and blue—are then used in the same manner, the examinee naming the color correctly as soon as visible to him. Yellow is frequently used to keep the examinee alert and is called a confusion color, but the extent of vision with this color is not recorded. A mark on the record with a pencil corresponding to the respective color indicates the point where the color is first seen.

The test object should be exactly 5 mm. in diameter, should be distinct and the colors standard. Certain defects inherent in the test objects in general use have been pointed out[17a] and new spherical test objects have been recommended.* (See Fig. 9.)

The eye which is being examined must not be permitted to shift the fixation from the white test object at the center of the arc. The test objects should be well illuminated. The light should be kept in the polar axis as much as possible. A small field is frequently obtained if the illumination is poor. A 200-watt daylight lamp gives a good, constant illumination, and is preferable to daylight unless strong north light is available, which is rarely the case. It must be borne in mind that the object will be seen before color is recognized. For example, red will appear black or yellow at the periphery of the field and will not be recognized as red until it reaches the red field.

Hertzog[66] believes that if pathology is suspected or exists, the campimeter (Bjerrum screen) should be used in examining the fields. Defects were found in the visual fields of forty flying officers who were examined by Doesschate.[46] The scotomata were mainly of the ring type; in only one was the ring complete. He found the ring 25° from the fixation point in some of these cases. He considers the scotomata due to the more highly refractile rays of light and similar to eclipse blindness.

The Effect of Altitude on Visual Fields.—The fields for form and color[12] have been taken in the low-pressure chamber at 5,000, 10,000 and 20,000 feet. Oxygen is administered when contraction is noted at 20,000 feet. To make sure that the changes are not due to fatigue, controls were made at sea level corresponding in time of day and time of interval to those taken in the low-pressure chamber. At 5,000 and 10,000 feet there is usually a slight enlargement of the fields for form and color; at 15,000, a slight contraction. At 20,000 feet the form fields of eighteen men showed a contraction of 14 per cent of their original size below,

*Test objects may be obtained from the American Optical Co., New York.

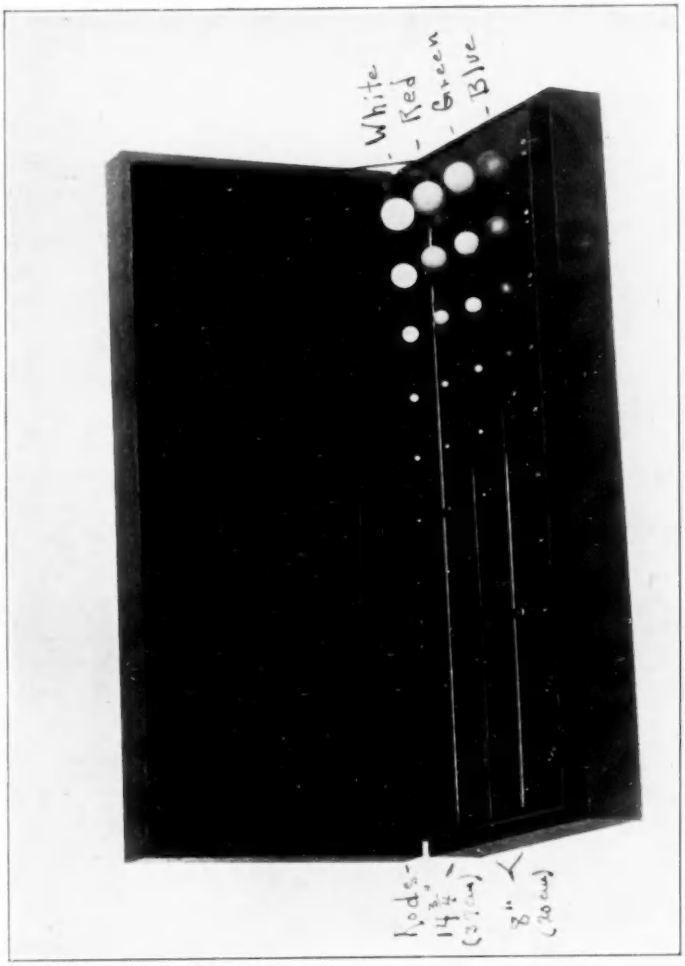

Fig. 9. New spherical test objects for form and color.

3.5 per cent in the temporal field, 4 per cent above, and 8 per cent in the nasal field.

Five minutes after returning to sea level, fields are normal in size. A return of the field to normal can be caused by administering oxygen, for four or five minutes, even at 20,000 feet.

Perception of Motion and Direction of Motion by the Retina.[13]—It is important that the aviator note the approach of an enemy plane before the enemy sees him; therefore, keen sense of perception of motion by the retina is a valuable asset to the flier. It was our endeavor to provide some method of taking and recording these fields in the hope that something of practical value might be found. We found that the field of motion is approximately 3 degrees larger than the field of direction of motion, which, in turn, is about 1.5 degrees larger than that of form.

Discussion.—It is important that the field of every aviator be examined with a perimeter. In examining for form it would be sufficient to use a 3/330 mm. white test object, and for color a 5/330 mm. red test object, maintaining 7.5 foot candles of illumination. For industrial, private and student pilots a four-point field would suffice, but for transport and limited commercial pilots an eight-point field should be required.

7. CENTRAL COLOR VISION.

The Value of Normal Color Vision.—Normal color vision[11] is considered important for the pilot by the majority of nations, and certainly it plays an important rôle in judging the color of fields and swamps in the process of landing. Good color vision is essential in distinguishing lights on airdomes and airplanes and in recognizing an enemy plane in time of war. Beyne[23] points out that it is necessary for a pilot when making a forced landing to have normal color vision in order to be able to differentiate, from a sufficient distance, the differences in the terrain.

Standards Set by the United States Department of Commerce.—The Ishihara or Stilling test for color vision is used by the Department of Commerce. If the candidate fails on whichever one is used, Jennings' or Holmgren's tests are given. The applicant will be rejected unless he is able to pass one of these tests. We believe that in cases of doubt the Edridge-Green lantern should be used.

Methods of Testing Color Vision in Other Countries.—The Edridge-Green lantern, Stilling's plates and Holmgren's wools are the tests most generally used in other countries. Onfray[82] suggests as an international standard that the examiner should

first test with the Ishihara and then with the Stilling plates. He believes that Holmgren's wools are not sufficient. In cases of doubt he advises Nagel's anomaloscope.

Normal vision is required by the British Air Ministry,[28] and Cantonnet[31] stresses the importance of having normal red and green color vision for landing.

The Effect of Altitude on Color Vision.—No change in color vision was noted in oxygen depletion experiments when using Stilling's plates.[11]

Discussion.—Many air pilots, who were known to be color blind, made successful records during the war. Experienced fliers may be able to compensate for the defect, and some countries do not require normal color vision for commercial pilots. In spite of this, however, it seems advisable to us to exclude men from aviation who have defective color perception.

8. LIGHT SENSE.

Normal light perception is apparently not considered important for aviators in the United States, as it is not required by the aeronautics branch of the Department of Commerce, the Army or the Navy. There is no doubt, however, that night flying and flying at dusk necessitates normal light perception. It is important that the commercial airplane pilot be able to adapt his sight rapidly. He must read his instruments, which are well illuminated on the instrument boards, and then immediately observe objects which are not well illuminated. It is necessary for him to have normal visual acuity in reduced illumination in order to perceive objects in the dark.

Apparatus and Methods of Examination.—Cantonnet[31] points out that it is not the light perception but night vision (visual acuity as it functions in reduced illumination) which should be tested. He believes adaptation in a dark room for eight minutes is sufficient allowance of time before applicants read letters, or preferably, the Landolt broken ring, at five meters. The tests are made through smoked lenses which have been determined experimentally to permit sufficient vision for a subject who adapts normally.

In addition to the original Förster photometer and the Nagel and Birch-Hirschfeld apparatus, practical instruments have been devised in this country by Cobb,[35 36] Derby[44] and Ferree, and in England by Spearman.[92]

International Standards.—Onfray[82] proposed to the Thirteenth International Congress in Amsterdam, in 1929, that light perception be tested by means of the Birch-Hirschfeld or Nagel apparatus, after five, ten and fifteen minutes of adaptation and that a curve of the rapidity of adaptation be attached to the application blank of each pilot. Furthermore, he believed it important that visual acuity at night should be equal, at least, to 5/100 for an illumination of 0.0015 lux after twenty minutes of adaptation.

The Effect of Altitude on Light Perception.—The effect of lowered oxygen tension on light perception has been studied[11] and also the threshold sensitivity for contrast with white and colored lights. Under the rebreathing test the threshold for light has shown an improvement in 25.9 per cent of the men examined; 44.5 per cent showed no change and 29.6 per cent showed diminished sensitivity.

In studying the threshold for colors, 71.4 per cent showed lowered threshold for red and green lights and 28.6 per cent showed no change.

Discussion.—The United States Aviation Service should add a test of adaptation to light and visual acuity in reduced illumination for all fliers, because one never knows when it may be necessary to fly at dusk or in darkness caused by a storm.

9. OPHTHALMOSCOPIC EXAMINATION.

The ophthalmoscopic examination is important in determining the existence of any intraocular disease, and especially in the elimination of applicants who might tend to have reduced visual acuity at night, particularly those with early peripheral changes of retinitis pigmentosa.

Commercial Standards in the United States.—The media, disk, iris, blood vessels, and retina of each eye[98] are examined and diseases and anomalies are disqualifying for all classes of pilots. Euphthalmine may be used.

The Importance of Using a Mydriatic.—For several years[12] we have stressed the importance of using a mydriatic when making a thorough examination of the fundus and determining the total refractive error in cases of aviators among whom we are to make a selection and to whom we are desirous of giving advice regarding the proper care of the eyes.

Discussion.—It is our belief that homatropin should be used and the fundus examined by the direct and indirect methods.

Retinoscopic findings at one meter should be recorded at the same time.

10. PSYCHOLOGICAL ASPECTS.

The personality of each candidate who applies for a license from the United States Department of Commerce[98] is carefully studied. It is certain that the psychology of the aviator will have an important bearing on his visual judgment and should always be considered in selection.

11. VISUAL REACTION TIME AND SPEED OF ADJUSTMENT.

No test is made of this important function by the Department of Commerce or the United States Army or Navy, although rapid reaction time is most important for the military and public aviator.

Discussion.—The more experience one has in examining the eyes of aviators the more convinced one becomes that the ability to react rapidly is of prime importance, especially for the military aviator. By the end of the war we[15] were devising tests all of which considered the time factor. Rapidity of reaction must be associated with sound judgment and accurate coordination. The importance of rapidity and accuracy in reaction time is stressed by Azzi[4] and Guillian,[61] Cantonnet[31] and Beyne.[22] The French authorities give the visual reaction time as 19/100 seconds. At Issoudun, France, in 1919, using the d'Arsonval chronoscope, the reaction time for monitors was 17/100, testers 18/100, students 19/100, French fliers 21/100 and fliers from the American front 20/100.

As an international standard, Onfray[82] proposed that the opening in the Landolt ring, 1/10 visual acuity, should be located in one second; the color of light determined in 0.25 second. It would be advisable for the Department of Commerce to add these tests to the present requirements.

III. THE EFFECT OF ALTITUDE, FLYING STRESS AND HIGH SPEED ON THE OCULAR FUNCTIONS.

The effects of altitude have been taken up under the various subjects, Visual Acuity, Accommodation, etc., and it is only necessary now to consider certain of the broader aspects of the effects of flying on the human organism.

Anderson[3] believes that greater attention should be given to the effect of the physical strain the aviator is forced to impose upon his body when flying—especially when attaining high altitudes. This writer points out that the increased speed of the machine,

the heightened ceiling, the rapidity which which the plane ascends, are all factors which have brought aerial warfare to a situation where the aviator is compelled to live and function in an atmosphere of diminished oxygen—the most vital constituent for life. Severe physical strain is added to the nervous tension.

We believe[14] that the causes of "staleness," other than the lack of oxygen, may be grouped under the term *"flying stress"* and that the low altitude work practiced universally in the last few months of the war was particularly trying. It consisted in "shooting up" trenches, marching columns and even soldiers in rest billets. This work was usually done at an altitude of 500 feet under conditions where an accident or a collision meant almost certain death to the aviator, as there was seldom sufficient altitude in which to right the plane. Low altitude trips in the absence of hostile aircraft are not so fatiguing, as the only strain is that of nerve tension due to watchful waiting.

IV. Summary and Conclusions.

1. Aviation ophthalmology is one of the most important branches of aviation medicine and requires special study, because the eyes and body are subjected to unusual conditions in flying.

2. Ocular assets of importance to the aviator are: normal and equal visual acuity, normal visual fields, keen perception of motion; rapid, simple and discriminative visual reaction time; normal near-point and rapid adjustment of accommodation; normal converging and diverging power, and rapid adaptation and good visual acuity in reduced illumination; normal color perception; normal resistance to fatigue and flying stress and exceptional ability to withstand the effects of altitude and lack of oxygen. The most important asset, however, is the visual training gained by experience.

3. Original examinations and re-examinations should be made with the thought in mind that carelessness, neglect or lack of knowledge may result in a fatal accident. Therefore, interest in the applicant must not be permitted to influence the examiner.

4. Certain eyes, especially those with muscle imbalance, convergence insufficiency, divergence excess, accommodative insufficiency and hyperopia, rapidly become inefficient under the effect of altitude.

5. It was found unnecessary to examine the eyes of aviators during the altitude tests because, after examining the eyes of 600

aviators, provided they were ocularly acceptable for the Air Service of the American Army in 1918, a mental or physical failure always developed before the eyes became inefficient from any cause.

6. We agree in principle that all eye examinations should be made by ophthalmologists, but in practice this is impossible. It has proved practical to have these examinations made by men who have had the training in aviation ophthalmology offered by the United States Army and Navy.

7. Because of the weakening effect of oxygen depletion and flying stress on visual acuity of men with ametropia, especially hyperopia, and the difficulties in landing experienced by men with low visual acuity, if their goggles become suddenly displaced or soiled, the present high standards for visual acuity set by the Department of Commerce should be maintained. However, the practice of considering the visual acuity of each applicant in the light of his experience and all other psychological, physical and ocular findings should be encouraged.

8. In judging distance, the aviator uses the motion parallax much more than the binocular parallax, but stereopsis is of great value in landing and in formation, stunt and combat flying, especially to the man untrained in using one eye alone. The Department of Commerce should maintain the present requirement of thirty millimeters, but judgment should be used in excluding men until the cause for poor stereoscopic ability has been determined and eliminated or stereopsis improved by orthoptic training. The present requirement may be unnecessarily strict for private pilots, especially in the light of the international standard proposed by Onfray,[82] but a waiver should not be granted before every effort has been made to improve the aviator's stereoscopic ability.

9. The Department of Commerce is wise in not insisting too strongly on normal muscle balance but in placing greater reliance on functional tests of binocular vision. There is no doubt, however, that men with heterophoria, especially exophoria and hyperphoria, do not, as a rule, withstand oxygen depletion and flying stress as well as men with orthophoria. Aviators with low degrees of heterophoria, if they have a sufficient reserve power of fusion, will seldom be troubled in flying.

10. In testing accommodation it would be advisable for the Department of Commerce to use capital letters which subtend

known visual angles. All classes of pilots should be able to read 400 millimeter print, with their distance correction if necessary, within a near point correction for their age, according to the standard set by Duane,[51] in 1912. If a disqualifying defect is found, the cause should be determined and eliminated and the near point brought within normal limits. If accommodation cannot be improved to normal the accommodative insufficiency should be studied in the light of all other findings in granting a waiver.

11. A normal field of vision is important for all aviators and this should be tested by the use of a perimeter. If scotomata or contractions are found in the visual fields, the exact cause should be determined before a waiver is granted.

12. Aviators should have normal color vision as long as colors are used for beacons, navigating lights and military markings by day or night. It might be advisable for the Department of Commerce to require the use of the Edridge-Green lantern when men fail to pass the tests with the Stilling or Ishihara plates.

13. Normal light adaptation and normal visual acuity in reduced illumination are important for aviators, and it would be advisable for the Department of Commerce to require the tests for light perception proposed as an international standard by Onfray.[82]

14. A cycloplegic should be used for all ophthalmoscopic examinations. The retinoscopic findings, as well as pathologic conditions of the fundus, should be noted.

15. Psychology is an important factor in visual judgment, and the individual psychology of the applicant should be considered in every ophthalmological examination.

16. The Department of Commerce makes no study of visual reaction time, although rapid reaction time for visual discrimination is important for the aviator. As an international standard, Onfray[82] proposed that the opening in the Landolt ring (1/10 visual acuity) should be located in one second; the color of a light determined in 0.25 second. It might be advisable for the Department of Commerce to add these or similar tests to the present requirements. Although Reeves' test for the reaction time of visual discrimination is probably the best, it would be impractical to use it except in certain large examining centers.[15]

17. The advisability of maintaining high ocular standards for aviators has been proved, even though these standards cause the rejection of 50 per cent of the men disqualified by the Air

Service of the Army and Navy of the United States and 70 per cent of the 7 per cent disqualified by the United States Department of Commerce.[39a]

18. The eyes of all aviators should be examined every month, but since this is not possible, the present standard set by the Department of Commerce is practical. No public pilot should be permitted to resume flying after an accident until he has been re-examined. All private pilots should also be re-examined as soon as possible after an accident.

19. The adoption of international visual and physical standards for aviators will do much to promote safety and to advance the science of aviation.

The research on the effect of altitude on the ocular function of aviators mentioned in this paper was done by Doctors Constantine, Sherman, Reeves, Dunnington, Skeel, Webster, Fitzgerald, Underwood, Goux, Stevenson, Hyde, Frances, Donnelly, Thomas, Rowe and Jackson.

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OTOLARYNGOLOGIC ASPECTS OF COMMERCIAL AVIATION.

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While the visual function is of paramount importance to the aviator, he must also possess good breathing space, good hearing and an excellent sense of balance. A full supply of air is necessary to the flyer, and in the thinned air of high altitudes his oxygenation is impaired, with danger to the accuracy of his responses in control of his ship. It is true that at such heights there is a long distance in which control of the plane may be recovered; but even a temporary loss of control may lead to fatal difficulties. This is especially true in flying high over the transcontinental routes across the Rocky Mountains and the Andes. Also, distinct effort is needed in expiration during flight, because of the high air pressure flowing past the plane at good speeds, due to the slip stream thrown back by the propeller as well as to the rapid movement of the plane. These matters are, of course, covered fully in the general medical examination, in the tests of breathing capacity.

For these reasons the otolaryngologic examiner is required to look very carefully into the matter of mechanical obstructions to free breathing. Septal deviations or thickenings; turbinal hypertrophies; nasal polyps and the underlying accessory sinus involvement; nasopharyngeal fibromata; adenoids; hypertrophied tonsils where there is a history of repeated severe attacks of inflammation; paralysis or stenosis of the larynx; nasal, pharyngeal or tracheal ozena with obstructive crust formation—all these conditions, if not easily remediable, require attention from the student flyer and must be done away with before he can be passed to the limited commercial and transport grade. In general, he must have at least four-fifths of the normal breathing space.

Septal perforation invites attention to the venereal history. Active syphilis and tuberculosis are causes for rejection. Any apparently syphilitic lesion, from the mucous patch to the old cicatrix, should invite serologic investigation; and chronic ulcera-

tive processes, suggestive of lupus or other tubercular lesion, indicate bacterial inquiry. Such persons, however, rarely present themselves for examination. Where old tertiary scars are found, positive rejection occurs. The department takes no chances on late lesions of the nervous system.

Occasionally cases of trench mouth, dental caries or severe pyorrhea require attention because of the danger of involvement of the tonsils or of the maxillary antra, with the possibility of bronchial changes. People who fly are likely to smoke a good deal off duty, and occasionally a reduction in nicotin will fix up a bad chronic bronchitis.

Hay fever of the pollen type is no bar to aviation; quite the reverse is true, for the upper air is relatively clear of irritating dusts. Nasal obstruction and asthma due to food and animal product allergies may occasionally require investigation.

Bad tonsils need to come out because exposure in open or bad weather flying is likely to stir up attacks.

Obviously also, the presence of foci of infection, such as apical abscesses, purulent sinusitis and tonsillar suppuration threatens recurrent "colds" as well as generalized febrile attacks with distant manifestations of trouble. Also, any malignant manifestation, and such deformities as severe cleft palate and harelip, are cause for rejection of the applicant. Former operative procedures on the larynx and trachea—as tracheotomy—will cause rejection unless it can be shown that the tracheal caliber is normal.

One who has traveled much by airplane is likely to wonder why good hearing is required. Certainly under present conditions an occupational loss of hearing gradually affects many pilots. Constant noise is one factor, and frequent changes of altitude another, which make for rapid deterioration in susceptible persons.

The student or industrial pilot is required to hear the whispered voice at three feet, and the transport or limited commercial pilot at eight feet, in either ear; the numerals 18, 23 and 66 must be used. The pilot must be able to hear his radio telephone signals and the shouts of his ground crew, copilot or passenger. His eustachian tube must be tested for patency, in the higher grades; temporary occlusion not only causes deafness but may condition severe dizziness during flight, particularly during rapid descent.

Acute or chronic otitis media or mastoiditis, with or without cholesteatoma, or unhealed perforations, are disqualifications; but

a trained pilot who has a mastoid operation may return to duty after a waiver of his defect upon recovery. Hearing in the combat services must be normal in both ears. Audiometer tests are not used in civilian examinations and are optional in the combat services.

External ear affections, such as cerumen or epithelial plugs, and eczemas, particularly that caused by the various molds or trichophytons, must be brought to the pilot's attention so that obstruction will not occur.

The vestibular function no longer holds the commanding position in aviation examination which it did during the war. Balance is, of course, constantly disturbed during flight, particularly in the rapid maneuvers of the combat plane. The air is full of currents, not only directional winds, but up and down currents and eddies. It is therefore necessary for the pilot to acquire the "feel of his seat," much as a good rider sits his horse, and he must be trained to interpret sudden movements of his plane, even in civilian flying, and to use the same technic which is constantly required in combat flight, in order to get out of sudden emergencies.

Vision has, as predicted during the war, turned out to be far more important than balance. The blindfold or fog-blinded pilot is helpless, while experiments have demonstrated that a deafmute with dead labyrinths can do pretty well in the air. Except in summer flight in an open cockpit, tactile sense is of little aid in getting the sense of position. Deep muscle sense—the feel of the body in the seat, the movement of the plane about the clothing—are of much importance.

For practical purposes, in commercial examinations it has been found that standing with the eyes closed, on one foot, with the other knee bent at a right angle, for three trials of fifteen seconds each for each foot, gives a very accurate determination of the candidate's steadiness. The turning chair is no longer used. Gait in walking must, of course, be normal, unless the result of a nondisabling fracture. Combat fliers must still take the turning test, with postrotatory nystagmus of normal amplitude and within normal limits—this because of the acrobatic character of much pursuit and combat training. No attention is paid to the minutiæ of past pointing and cerebellar orientation in the commercial pilot, and the experience of several years has justified the abandonment of this time-consuming testing. In fact, the expert pilot will not

past point normally; he "accommodates" to the unusual motion because of habituation.

Facility in adaptation to rapid position change is obtained in military aviation by the use of the Ruggles "orientator"—a cockpit mounted on universal pivots, and motor controlled so that it may be thrown into any desired maneuver by the pilot or his teacher. The green pilot shows all the classic signs of labyrinthine sensitiveness at first, but speedily overcomes them. The hypersensitive individual, because of added emotional stress under so-called "air sickness," is a worse risk than the person with diminished labyrinthine sensations.

The semicircular canals are not alone in maintaining proper vestibular balance; we must not forget the static effects of the otolith mechanism stressed by Quix, with the pressure upon the cristæ in the utricle and saccule. The Bárány chair tests only the horizontal rotations, while much of the flier's work, especially in combat training or in landing and taking off in fast planes, is more or less in the vertical plane, not laterally at all.

It should not be forgotten that psychic conditions may greatly alter vestibular sensitiveness. Worry, fear, excitement over the outcome of the examination, the aftermath of a "wild party," sexual excess, may interfere decidedly with the showing made, as well as with the pilot's behavior under training. No pilot should be trusted in a plane with his ears buzzing with alcohol, and government regulations are very strict in this regard.

It must not be forgotten that muscular imbalance of the eyes may cause vestibular symptoms under flying conditions—an added reason for care in the eye examination. Other sources of labyrinthine irritation must be eliminated: high blood pressure, excessive smoking, apical abscesses, chronic sinusitis, gall bladder disease, intestinal stasis. Syphilis is a permanent barrier. Locally, the chronic suppurations, especially with high perforations and cholesteatoma, and crusting in the radical mastoid cavity, cause rejection of the applicant.

The American pilot is less exposed in the modern plane than he was a few years ago. He has learned to keep cotton in his ears or to put a powder puff in each side of his helmet. Also, he has a windshield, and in the larger planes a relief pilot; and with improved instruments, stabilizers and the radio telephone his lot is less dangerous and far less trying, emotionally and physically. In general, he has far better equipment to fly with than has the

European pilot; his engines are quieter, and his plane is more modern. New planes in Europe appear mostly under military and naval control; the commercial fleets are old, as time goes in aviation.

The requirements of the International Commission for Air Navigation, which controls most of the air traffic over Europe and Asia, are equivalent to those of the Hispanic Air Union. German and Austrian pilots, while not under the Commission, adhere to similar standards, as they are constantly landing in Holland, Czechoslovakia and other countries. Private pilots must hear the whispered voice at one meter, while transport pilots must hear the C forks of 64, 256 and 4096 d. v. at 1 cm. from each ear for the normal period of time. Eustachian tubes must be patent and the vestibular system free from irregularities or abnormalities, and no serious nasal or buccal disease is permitted.

Because of military necessities, European pilots are rather likely to be found in the army or navy reserves. In France the rotating chair is always used, ten turns in twenty seconds, right and left, head 30° forward. In England it is used only as a check up on those likely to go into acrobatic flying. According to the most recent circular of the Royal Air Force, "as a result of the examination of first grade pilots by the Bárány tests, it was found that the reactions to rotation might be excessive in some individuals and diminished in others, but in neither case did the reactions of the vestibular apparatus seem to indicate an airman's probable flying ability."

The French examination is very thorough for the beginner, but rather perfunctory for the experienced pilot. Blind walking, forward and back, and the Romberg with one heel touching the other toe, are added to the routine turning test. One has the feeling, watching the European examinations, that the possibility of military service lies back of the careful records which are kept at Le Bourget and at the Air Ministry in Whitehall. Similarly patriotic care should actuate any American physician who is charged with such duties.

806 Medical Arts Bldg.

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THE PRESENT STATUS OF AVIATION IN THE UNITED STATES TODAY.

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It is a great pleasure for me to be here tonight and have an opportunity to talk to you about aviation, my pet hobby and present vocation. And I am particularly glad because of my firm conviction that medicine has a very important part to play in the development of aviation.

As far back as 1916, say about the end of the second year of the World War, the Allies, and undoubtedly the Germans too, became aware that accidents were taking a far greater toll among their aviators than anything else. Investigation showed that roughly 90 per cent of the fatalities were caused by the pilot's error, 8 per cent by material failure, and only 2 per cent by the activity of the enemy. In other words, the loss of personnel was occasioned not by the actual fighting but rather by the training and incidental flying. Therefore a study of the personnel was begun, and physical examinations were introduced by Great Britain, Italy and France. Shortly thereafter the United States followed suit, and since then physical examinations have been required. In recent years, in addition to physical examinations, psychological examinations have come into being.

Physical examinations remove a great number of candidates from the field of aviation. For instance, of our Annapolis graduates, who have, of course, been subjected to weeding out by physical examination on entering and while at the Naval Academy, approximately 50 per cent are unable, because of their physical defects, to pass the requirements. Examinations are held annually, and gradually we have also been establishing flight surgeons at every naval air station and with every air group at sea. The flight surgeons live in company with the aviators, continually check up on them, and assure themselves of their well-being. Where the flight surgeons are unable to see the boys daily, weekly tests are given to guard against mishaps.

The eyes are of great importance in flying, but rather than any one particular thing it is the general health of the entire body that is important, for the increased altitude and continual change in altitude, as well as the nervous strain in general, require that the body as a whole be in excellent shape. The increased altitude, of course, lessens the oxygen and places a burden upon the heart and all of the organs of the body. For example, from a layman's point of view, it is about as much of a strain upon a human being in a general way to fly for a couple of hours at ten thousand feet as it is to play 18 holes of golf.

The study of psychology in this connection has been most enlightening, and for several years now we have been learning much in this field. In a group of approximately 682 candidates, examinations have been made and these men divided into groups of good, bad and indifferent. The results of these tests have been extraordinarily successful from the point of view of the psychologist, and it is felt definitely that those who passed the examination as "good" will assuredly be acceptable aviators. Those passing badly will not make successful aviators, while the group in between the "indifferent" ones, should be given the actual practical flight training to determine their fitness. In other words, we believe that we will be able to eliminate a certain percentage of the personnel through this process at the very start and we will be able to be assured in regard to others. The cost of training men makes it very important to be able to eliminate those whose training would be in vain. We are now almost sufficiently satisfied to begin the practical use of these psychological examinations in weeding out our personnel.

In connection with naval aviation, the department has compiled pictures of the activities and maneuvers during the past year. These despite all phases of flying with out Fleets, and I hope that it will be of interest to you.

(Movie.)

I want to thank you very much for letting me talk to you and show you what we are doing in the air in the Navy.

THE TEACHERS' SECTION.

PURPOSE OF TEACHERS' SECTION.

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The American Academy is the largest special society, both in membership and capital, existing in organized medicine in this country. It is universally accepted that matters of interest to Ophthalmology and Otolaryngology are of interest to the Academy.

It is encouraging to observe the rapid expansion of Academy activities. Sound judgment, however, directs that care and thoughtful analysis must be given to suggestions for further expansions, yet there remains the obligation of the Academy to its members, and the implied obligation to maintain a common sense scientific balance between facts and fancies in matters wherein the specialty is concerned. Therefore, final acceptance of a suggestion is determined only after careful deliberation, and only after clear cut objectives have been established.

The Council has been instructed to organize a Teachers' Section. The proposal is herewith submitted to the membership for deliberation.

I have been requested to outline what might be construed as objectives for this proposed section.

The evaluation of the art of teaching medicine, viewed in retrospect, constitutes one of the interesting chapters in the history of medicine. From the era of fads and foibles to Hippocrates' magical formulæ, to the diversified scheme of present day medicine, have been but steps up the ladder of progress. Even as the teachers of yesterday labored, each in an environment peculiar to the times, we who teach today find ourselves subjected to that ever present and ever annoying halo of standardization. That standardization construed to its ultimate dimensions is perfectly applicable to matters executive is acceptable, but I question the

possibility of ever bending that higher type of good, sound teaching to the iron rules of any universal standard.

Attitudes, traditions, localisms, always have and probably will continue as factors in appraising any motive or motives actuating the theme of teaching in a definite field or region. Underneath the obvious or apparently obvious reasons are causal beliefs and habits, forces that determine factors in teaching policies as economic factors determine finance in industry. Therefore, I believe standardization, in the strict sense of forcing methods or rules, should not be an aim of this proposed section.

Recently Duke-Elder, quoting Professor Henderson of Yale, defined scientific investigation as "To understand—to predict—to control." Of these, the third is the last to be attained, and the greatest, for unless one has first gained an understanding of the nature of basic phenomena, prediction is inaccurate and control impossible. I sometimes feel that in the days of yesterday better common sense medicine was taught than in certain places now. True, the theme was clinical medicine, empirical perhaps, but yet clinical medicine. Curiously enough, with the acceptance of the Flexner survey, improvement was noted as has been definitely observed following the activities of the American Medical Council on Medical Education. That medical schools are more nearly functioning along unified lines in recent years can undoubtedly be accepted as in a large part due to the influence of the American Association of Medical Colleges. These forces have expended their energies largely along executive lines and intradepartmental relations, having only indirectly affected interdepartmental activities.

Perhaps a Teachers' Section can find its objective in carrying on from where these forces have stopped.

Dr. Meeker, Dean of the Graduate School, University of Pennsylvania, recently made the significant statement that well organized surveillance, directed from within a specialty, would be gratefully received and would wield sufficient influence to be materially felt in any graduate curriculum.

An analysis of the undergraduate school reveals a wide discrepancy in the principles taught. It is interesting to chart the variable ideals seemingly running through schedules and from which to construct a sound, logical, comprehensive outline of the general medicine of our specialties would indeed be difficult. Assuming, of course, that only the general medicine of ophthal-

mology and otolaryngology belong in the undergraduate curriculum.

Reflecting for the moment upon the changes noted in the medical curriculum during my own generation, it has been plainly obvious that a continuous development process has been taking place, evidently subject to forces without the control of any one individual or group of individuals, but that have, nevertheless, in a directional way, been influenced through sound academic and logical reasoning in most instances developed independently and with but little cohesive effort from a national source. Perhaps a Teachers' Section would materially assist in stabilizing facts and eliminating fancies.

In visiting the Graduate Schools, I have had this peculiar reaction and have wondered if you (the other fellow or myself) were boss, would we hire ourselves. If before answering this question, five minutes of honest thought was given to its significance, it is believed that 99 per cent of us (the boss included) would give negative answers. This is but another way of repeating the age old truism "that no honest man can gaze into a mirror, look at himself intensely enough, and not blush."

There must certainly be a minimum schedule of hours, a minimum curriculum, the observance of which will turn out students properly founded in the basic sciences of ophthalmology and otolaryngology. Equipped so he could honestly be certified to the public, to the profession, as a safe man in the specialty.

I have no wish or intention to depreciate the art of teaching ophthalmology and otolaryngology, for of all the branches of medical science, I feel that none can claim proselytes more devoted or more brilliant than many laboring in these specialties, yet viewing the situation as a conglomerate picture, it would seem that most of those professing responsibilities have been merely skirting around the fringes of their essential problems, studying gross clinical phenomena, attempting to control without understanding.

May I, with some trepidation, state that the purpose of a Teachers' Section of this Academy probably would be to understand the requisites of our specialty in its adaptation to general medical science? To construct a minimum level of knowledge necessary to acceptance as qualified applied to the individual, to the undergraduate or graduate school.

Moreover, new ideals are constantly being forced into the medical curriculum, and I am sure that a carefully studied expression from this body upon matters of interest to us would be seriously considered by those in control of teaching universities. Perhaps the most recent suggestion is the experiment now being used at Tulane University, where a transfer of emphasis from didactic to the ultra-clinical medicine has been made, which, if accepted, will revolutionize our present day methods.

May I close this part of my discussion with the rather frank statement that the philosophy of teaching can only be acquired through contact with others similarly interested.

Should it be the desire of those assembled to formulate a Teachers' Section, I am offering the following suggestions, simply as an outline for discussion, out of which can be formulated the petition to the Council:

PLAN OF ORGANIZATION.

That the section be divided into four subsections—

A. Graduate Ophthalmology.
B. Graduate Otolaryngology.
C. Undergraduate Ophthalmology.
D. Undergraduate Otolaryngology.

That an executive committee of five be appointed by the Council for a variable number of years, from one to five, and that hereafter one fellow be elected each year to replace the fellow retiring. That the first four years be served as leader of the subsection from which he comes, and his fifth year of office be as General Chairman.

That the executive committee construct the program for each subsection and the general section meeting. That the General Chairman become ex-officio member of the Program Committee of the Academy. That the executive secretary of the Academy be the secretary ex-officio of the section.

That members be elected to the section from those fellows of the Academy holding positions in acceptable graduate and undergraduate schools, and those conducting residences in ophthalmology and otolaryngology acceptable to the section.

That the Council be petitioned for a grant of $500.00 annually from the research fund, as a working capital.

That minimum dues be charged each member, which will help defray expenses and out of which the cost of an annual dinner may be taken.

PROGRAM OF ACTIVITY.

Feeling that the Section should confine its activities to the intelligent use of statistics, the construction of acceptable policies from intelligently interpreted facts, it would seem fitting that commissions be appointed to gather information from universities and those interested in teaching and, after collaborating the material, present it to the proper subsection for discussion and approval.

I would suggest that each subsection meet separately to discuss its own problems and that the latter part of the session be a joint meeting, following an annual dinner. A nationally known educator could be invited as a guest speaker for the dinner.

SUGGESTIONS FOR COMMISSIONS.

The undergraduate curriculum in ophthalmology.

The undergraduate curriculum in otolaryngology.

A statistical study of clinical work in teaching hospitals.

A survey of the basic sciences of ophthalmology and otolaryngology as developed in the inter-related departments, namely, anatomy, physiology, pathology, and bacteriology.

A minimum graduate curriculum in ophthalmology.

A minimum graduate curriculum in otolaryngology.

A minimum curriculum for residencies in ophthalmology.

A minimum curriculum for residencies in otolaryngology.

Comparative studies of graduate courses conducted in America and abroad.

Innumerable thoughts develop, each of sufficient merit to warrant investigation, and I can visualize increasingly important work as the section progresses. I know of no organization that has the available personnel, the machinery to do with, such as this Academy. I sincerely believe that a section, correlating ideas, a coöperation of minds, trained to scientific interpretation, could do for ophthalmology and otolaryngology, in the pedagogic field, that which would be impossible in an individualistic way.

To clearly define specialism is the uppermost thought in current medicine. This thought, elaborated to the establishment of peda-

gogical formulæ for our particular work, is an ideal to be seriously considered. The development of a Pedagogical Research Section, if you wish.

If a Teachers' Section is created, its policies, program of activities and objectives, must be entirely different from those accepted for other sections. Overlapping of practices and purposes between sections should not only be avoided, but can be fatal to the organization as a whole.

PROBLEMS OF GRADUATE TEACHING.

DUNCAN MACPHERSON, M. D.,

NEW YORK.

A problem of graduate medicine, particularly to us, that of otolaryngology and ophthalmology, is the realization on the part of leading and influential otolaryngologists and ophthalmologists of the necessity of nationally organizing and then proceeding to take national action toward improving our schools.

In all departments of postgraduate medicine we need schools that will prepare men to give the best possible service to the public.

What are our outstanding defects and what plans can we devise to overcome them? After contacts with young and older men, prospective and practicing specialists, keeping my ear to the ground for reports from many men who have traveled at home and abroad, I believe I am justified in drawing the broad conclusion that there is no one properly qualified training place in the world meeting the demands of our men honestly trying to qualify for practice in the sphere of otolaryngology, barring, perhaps, a few in large hospitals where internes are taught basic subjects in addition to routine handling of patients.

Our American schools are unable, one from one deficiency, another from some different cause or causes, to give courses that the teaching staffs consider desirable. European schools, while highly advertised by themselves and by some of their students, are far from satisfactory to our men who go abroad. I have been told by many enthusiastic, good students who went abroad after training here and knowing what they needed and what they failed to obtain here in America that disappointment met them in Europe.

We must coöperate nationally, allowing enough accommodation and pliability to permit thrift of individual initiative among institutions. Our problem at present is not so much the detail of curricula. That will follow if we can successfully plan, direct and regulate teaching institutions throughout the nation. It seems to me that we have been working on details while we neglected

fundamentals. We have superstructure without appropriate foundation. Each school runs its own show, much as undergraduates did, until forced to do otherwise. No further elucidation of the point I wish to make is necessary before this meeting.

Whether more men than needed are training for and practicing otolaryngology and ophthalmology is important information that we need. (I believe that up to two months ago about 40 to 45 per cent of those expected to attend the Congress of the American College of Surgeons in New York were ophthalmologists and otolaryngologists.) What facilities have we to offer in the way of internships and what kind of training are we giving to internes even in the best hospitals? Should we encourage prospects to carry on as general practitioners for a given period before admitting them to the specialties? At what age does the average man fail in potential proficiency for adapting himself to a new sphere of medical practice and what help may we hope for from the already hard pressed, overcrowded curricula of the undergraduate schools? What model of teaching should be accepted as a minimum for preparing men and how are specified requirements to be enforced? In former years I undertook to outline in a very general way a prescribed minimum course of study in otolaryngology. Such a course sketched intensive work under individual instructors covering something like 1200 hours for a period of eight months—i. e., an academic year—but by coöperation of our leading men and schools, amplified by persuasive coercion, I believe we could induce enough schools to require twelve months in otolaryngology and that enough students would enroll to meet the public requirements. Overenthusiasm for preparing men for the specialty must not be allowed to blind us to the necessity of providing places for graduate study where general practitioners may learn how to meet the emergencies consistent with their environment. Traveling clinics do not lend themselves satisfactorily to our work, even to the skirmishing of the general practitioner. Certainly the graduate leaves his alma mater innocent of necessary otolaryngological equipment, but that being true of other specialties, the otolaryngologists are not in a unique position. Convincing the undergraduate teacher that otolaryngology and ophthalmology are no longer to be categorized in the minds of his students as minor specialties would indeed be a major achievement. Through this, once true, true no longer but persisting habit, the doctor carries

through his pre-otolaryngological and ophthalmological life the belief that he can at any time, with very little preparation, while young or old, become a competent ophthalmologist or otolaryngologist, and thus are the undergraduate schools unwittingly spilling over into our specialties disproportionate numbers of incompetents and mediocrity, adding difficulties to our attempts at control. But what can be expected when men high up in undergraduate schools who, owing to the way medicine has unfolded, hold the attention of lay contacts, have the ear of universities, the trustees and the financiers interested in medical work, ask "Is there any field for postgraduate teaching?" Undergraduate schools need enlightenment, and we sorely need their favorable coöperation enlightened. Graduate medicine, however, is surely showing a convincing capacity for greater and greater appeal. There is no such thing as a minor specialty in medicine. In fact, the surgery of otolaryngology is so fraught with high mortality that the general surgeon now deems it worthy of his active participation, and our brightest young surgeons need no longer avoid the battleground of otolaryngology owing to lack of opportunity to show their dexterity because of surgical safety as evidenced by a low death rate. Undergraduate schools should inculcate in the minds of all their students the necessity of continuing their studies after graduation.

Graduate medicine deserves financial support. Its needs last during the lifetime of the medical man, while undergraduate teaching lasts but for a few years. However, we must not be discouraged. Einstein gets front page publicity though but twelve people understand him. Parenthetically let me say that the efficiency of the undergraduate school in dealing with the complexities of its jammed curricula compels our admiration, and their courses are doubtless judiciously balanced. Graduate and undergraduate schools believe in evolution and each must bear with the difficulties besetting both.

Shortcomings lie in inferior graduate schools largely due to lack of their inspection and a method of compelling them to deliver the courses prescribed and advertised, unfilled need of central bureaus where prospective students may obtain reliable information and the need of qualified teachers, some of whom should be full time men, consequently necessitating schools with good financial backing (for no school can furnish the best equipment and teachers if dependent upon fees of students for income). Many teachers

should be men who are practicing their profession among the people and not in the wards only, and such teachers are not as numerous as needed. I doubt that 15 per cent of available practicing otolaryngologists in New York would affiliate with a teaching school in preference to a non-teaching institution. A teaching institution requires men who will study and men who can and will conscientiously cover the prescribed hours of work. Students sacrificing time, money and family should not be expected to be satisfied with clinics beginning and ending from ten to fifteen minutes short of prescribed time. We need schools where advanced laryngologists may select work that they know they need, and I am hoping to see such a school developed. In fact, I expected that Dr. Dean would have introduced such a one ere this.

Applicants should be sifted. Certain ones are not fitted for surgery. Schools should be inspected by men appointed by the different national societies. Information given by institutions in response to the ubiquitous questionnaire is apt to take the form of submitting their advertising matter, and I believe there is evidence to show that schools do not always render unto Caesar things that Caesar has a right to expect, judged by the infidelity of the written report.

Medicine suffers from the overproduction of printed matter, misnamed literature. Much harm is done by publication of inferior material, compilations and duplications and unworthy exhibits. It is to the advantage of all of us that we keep informed regarding the advances and work carried on in the medical world as a whole, but how is it possible for any medical man to keep reasonably abreast of important advances when he is forced to pick and cull a small percentage of original from pages of poor imitation? One of the inevitable evil effects is that none of us can cover a reasonable amount of the printed matter turned out. Most laryngological literature, so-called, goes unread by members of other medical departments, as evidenced by the advice given by the profession to their patients where rhinological and laryngological direction is necessary. By an excess of literature and its careless selection we are all losers—always excepting those who thrive thereby economically. We fail to recognize the good work of other departments because of ignorance due to redundant fertility. I am reliably informed that certain young men believe it commendable that they contribute by a self-imposed schedule so many papers per month or year. We hear today many stories

of ovariotomies done in years past. How many of these were the result of redundant literature, and how many patients are robbed of rhinological attention because medical confreres see so many operative failures resulting from unsophisticated belief in the printed word? Economic success confers a certain immunity to criticism, unfortunately.

Are our standards of living so high as to be undermining our economic morals? Is education, like scientific inventions and discoveries, moving faster than our adjusting capacity? On the literary front our editors must be our sentinels.

I believe it would be easy to bring up to date information regarding the capacity of institutions that might be ranked as class A for internships and ascertain wherein they might yet further improve their facilities, discover for what number of students other provisions should be made and devise methods for meeting the situation. Necessary information should be given willingly to designated agents of the national societies by institutions, whatever their standing. Institutions refusing to meet the minimum standards would soon fall in line when the central bureau of information failed to endorse them and when their product failed to rank as the peer of a competitor.

For several years some of us have been advocating dividing the North American continent into districts modeled after the plan of the Triological Society and appointing men to report to a central bureau the work carried on by schools in any particular district. One of the great difficulties outside of institutions where satisfactory internships are available is to overcome the inability of teaching major operating on the living. To overcome this we began a survey of the American hospitals where we might hope to place, for a shorter or longer term, men who had spent the minimum time qualifying—smaller hospitals where men could at least act as chief assistant and observe at close range a sufficient number of major operations before going it alone.

Many of our teaching problems are met by affiliation with a university where its chemical, biological, physical and other departmental facilities related to medicine may be drawn upon, but generally speaking, I believe the departments of graduate and undergraduate medicine should be entirely independent the one from the other. Neither understands the problems of the other. The graduate teacher has this in his favor, that he has been through

the mill of undergraduate work, but the dogmatic expounding of the undergraduate teacher has little place in the mind of the graduate of a few years back who since graduation has learned to doubt some of the positive virtues claimed for strychnin, digitalis, climate and bitter tonics. He is more or less a skeptic, and the undergraduate teacher does not recognize his child and has little patience with him. Graduate teaching is in itself a specialty.

In writing this paper I purposely avoided the discussion of details. Our present task is to develop a national plan dealing with generalities to which for endorsement all schools with a moral sense could and would subscribe. This would in no way interfere with the work of men who confine their energies to highly specialized courses. The short course in otolaryngology, frequently resultant from honest attempts to meet brushing-up requirements of the general practitioner, can easily become injurious. In my own school, founded 50 years ago, for so-called brushing-up courses in all departments of medicine—we were so successful in the brushing-up business in laryngology that before we realized it men who took our three to six weeks' courses in otolaryngology were broadcast throughout the country practicing as specialists. Brushing-up courses should consist of diagnostic work. Ability to open an ear drum or peritonsillar abscess should be part of every man's armamentarium. But I feel that it is a public danger to indiscriminately teach such applicants how to remove tonsils and adenoids, do septal resections, etc. Occasionally, in outlying districts, the general practitioner may find it necessary to perform tonsillectomies and adenoidectomies. A compilation of so many beds in such and such a city is useless. We need teaching beds.

It is true of graduate medicine as it is true of all educational departments in a university that the professional staff and the economic powers, with which latter rest ultimate decisions, are too remote the one from the other. The trustees may know little of the ideas and aims and ideals of teachers from whom should spring at least the academic, and yet this entrusted power represented by the financial body rarely meets the teacher.

The necessity for action in this field, in addition to what is being done by present organizations, is quite evident, and all influential teachers should be invited to coöperate. It is claimed that there are already too many organizations and meetings demanding attention, but how many is too many? I believe that was a

discouraging note struck before the birth of the Triological and possibly of this organization. So long as there is demanding work, new or old organizations must do it. It is not necessary or advisable that those who consider themselves already overtaxed should be conscripted for the essential work. In fact, pioneering of any kind requires enthusiasts for workers, but advice from the wise, even if they are lukewarm and on the side lines, often results in advantages to both.

STANDARDIZING GRADUATE COURSES IN NEW YORK CITY BY THE NEW YORK ACADEMY OF MEDICINE.

S. J. Kopetzky, M. D.,

New York City.

It has been my privilege to serve on the Committee on Medical Education of the New York Academy of Medicine for a long time and to be a member of its Executive Committee. I have heard many discussions similar to Dr. MacPherson's. I have heard many arguments about standardization and its desirability and undesirability in teaching. The Committee on Program here wants me to present to you the activities of the committee in New York with reference to the teaching opportunities offered in both the graduate and the undergraduate, but more particularly the graduate, educational field in New York City, under the supervision, if you will, of the New York Academy of Medicine.

It seemed to us that there are many courses offered to visitors in New York City that had no right to be given. Dr. MacPherson has told you of one and our committee made many similar observations. Accordingly, a bureau was established and all the teachers in New York in the special branches in which we are interested undertook to submit to a mutual checking up. This checking up was not done with an idea that the teachers' individual methods should be standardized or made to conform to any one measuring rod. There has never been any attempt to standardize, and I believe with Dr. Wherry that no such attempt should ever be made. There is something in the individuality and the personality of a teacher, something in the manner and quality of teaching, that you cannot standardize. You can standardize the hours you want a man to devote to his course, but we have not even attempted that in New York.

We do visit each other, and that can be done under the arrangement outlined by Dr. Wherry. I am not on the staff of the Postgraduate School, but it has been my great privilege to be a visitor there in the Otolaryngological Section on a number of occasions; similarly, Dr. McCoy, my associate at the New York Polyclinic,

has been a visitor at the Manhattan Eye and Ear Infirmary. In the same way we have received visitors from the Postgraduate School. Usually these visits come on very short notice: the visitor walks into the classroom or wherever the students are at work. If he can give any constructive criticism he does so then and there and the suggestion made is discussed by the two men, both working in the same field and both interested in the same idea of elevating the standards of graduate teaching in the City of New York. Usually, both men are Fellows of the Academy. Very often their departments are represented on the Committee on Medical Education of the Academy.

These methods have resulted in the elimination of fly-by-nights in the city. I will pass around a little booklet showing the activities of the Committee on Medical Education. There is no attempt at dictation; there is no attempt to formulate a fixed teaching schedule,—to say that a course given at the Postgraduate School must exactly parallel or follow the teaching methods of courses at the Eye and Ear Infirmary or at the Polyclinic. Nevertheless, since the institution of the procedure which I have outlined, there have been no complaints from the students who come to take the courses.

Dr. MacPherson said that they have discontinued short-term courses for a while. As soon as Dr. McCoy and myself came in charge of the department at the Polyclinic, we abolished them entirely. The type of men who come to study wants the type of course we now give. I do not know whether that is the case at Postgraduate. We require certain qualifications of our applicants and we have eliminated short-term work entirely. Our men take a year or more. They must stay a year; and some stay longer.

I do not know what else the committee wanted me to tell you. We have an approved list in ophthalmology and an approved list of courses in the basic sciences that we recommend to all students in ophthalmology and otolaryngology as a preliminary.

Whether or not the American Academy approves the Teachers' Section, I have heard no scheme or outline as good as that sketched in the brief paper that Dr. Wherry presented this afternoon. Eventually such a body will be established, because those of us who think in terms of teaching and in terms of the student know that it is necessary, and when a necessity becomes urgent enough it results in action somewhere. The necessity which Dr. MacPherson has shown is a necessity of which I and many others

are convinced. To me, it seems that the American Academy is the obvious body to work out an organization such as Dr. Wherry has outlined, not with the idea of controlling or standardizing teachers, but to afford a center for the discussion of the difficulties encountered by graduate teachers and to formulate plans for the perfection of graduate medical education throughout the United States. The scheme that we have heard today seems to me to be the best thing that has been suggested to meet our needs. We have no precedents and we must work cautiously but progressively to establish the thing we desire.

DISCUSSION.

DR. BURT R. SHURLY, Detroit: It is a well known fact that for many years every educational problem that has progress in it has progressed by virtue of the strides made in teaching. In our own Board of Education in Detroit the progress that is made in uplifting teaching comes almost entirely through the meetings of teachers and principals and those who are interested in all the various kinds of teaching. Without these conferences the progress would not amount to much. As I look upon it, this is a conference of teachers with the idea of uplifting, of constructing something better. It is a new movement that has in it everything that is good and is certainly worth a trial, because in all the history of medicine we have gone on teaching year after year without much consultation—it has been almost an individual matter—talking perhaps with one or two men in our own particular environment. So the need of this program is exceedingly great, it seems to me.

I have served for a long time on this Board of Examination and have examined more than two thousand men who come up from every variety of school, and when you look over a cross-section of these men in the various parts of the country it is interesting to see that in some centers there is almost complete failure, and in others there is brilliant success. But I think this organization of teaching would bring about a better understanding of what is the ideal in undergraduate teaching and in postgraduate work. I feel very strongly that there should be and must be certain centers where postgraduate education will receive complete co-operation and help, so it will become stronger and better, because only a few centers such as we have in our large cities have developed. Not so long ago we thought it necessary to go across the water to get a certain amount of education to carry on the things we wanted to do. Now that is not necessary. If this organization would do nothing more than to give stimulus and enthusiasm and enable us to concentrate along the line of better teaching, concentrate our postgraduate work in certain centers, I think it would be worth while to give it a good trial and see if there are not enough of our men who are willing to give time and energy to making their subject vital, so that these men may receive the highest type of teaching.

DR. JOSEPH C. BECK, Chicago: Perhaps some of the gentlemen present will recall a symposium at the Triological Society a number of years ago, in which Dr. Wm. L. Ballenger and some others participated, on undergraduate teaching as it was practiced in their particular schools. It was a very interesting discussion, but it stopped just there—with the discussion.

I am enthusiastic over the report of Dr. Wherry and shall support it to the best of my ability. In my school, the University of Illinois, we do not have postgraduate teaching on account of the financial conditions. It will not be supported at this time because they say it is too expensive; that in order to make it a good course they would have to have the fundamental branches, which makes it impossible.

The discussion here has been very constructive, and if a section is formed in this great Academy, subdivided in the way Dr. Wherry has pointed out, I believe it will bear fruit.

DR. MARTIN COHEN, New York City: With respect to the proposal of Dr. Wherry, I am strongly in favor of the resolution.

For the past fifteen years I have been instructing students in ophthalmology at the New York Post Graduate School and Hospital.

We divide our course of instruction into two main divisions, viz., those who desire to follow, later, the combined practice of eye, ear, nose and throat, and those who desire to follow solely the specialty of the eye.

The first group, we offer a basic course in ophthalmology for four months; its schedule can be procured from the dean of the school.

The second group is divided into three divisions: First—The basic ophthalmological course. Second—A four months' eye externship. Third—A four months' eye internship.

After the year's completion of this course under school supervision, we advise still another year in our institution in advanced practical and theoretical work, or else a year or more in some recognized approved eye institution.

If Dr. Wherry's concise and remarkably well presented proposition be acceptable to the Council of the Academy, I feel confident its effect will result in the universal recognition of a proper standard in teaching.

I endorse the suggestion that a recognized board be appointed to recommend the justification of the candidate's ability to practice his specialty.

DR. PERRY G. GOLDSMITH, Toronto, Canada: I was very glad to hear Dr. Kopetzky say that he has revolutionized the teaching of otolaryngology in New York. Lord knows it was needed. I am very glad they have had the courage of their convictions and have swept out these smaller institutions.

My work is teaching undergraduate students. I am to do that according to the instructions of the faculty. The faculty control the number of hours, and my job is to help the faculty turn out well rounded general practitioners, not specialists.

Teaching in Toronto University consists of demonstrations in anatomy and of the method of using instruments for simple examination of the normal individual. That consists of about twelve hours. That is, in the year before the final year. Next year they are taught at the out-patient clinic

to use the methods of examination with which they are supposed to be familiar, and only patients with simple ailments are demonstrated. No students are taught operating; they do not see operations. They are simply taught ordinary general practice. It is pointed out that their responsibility to the public is such that she expects them to advise wisely as to what is best to do in the ordinary case of ear, nose and throat seen in general practice. They have one textbook, viz., Logan Turner. Large textbooks are of little use to undergraduates. At the end of twelve months they come up for examination, and the more they know of general medicine the better they stand the examination.

We put out a series of questions two weeks before examination, and they are sufficient to entail their reading the textbook. I will give you a few to show the type of question. The examination is oral, not written, and includes the methods of using the commoner instruments. "What is the cause of enlargement of the cervical glands?" They must include in that not only Hodgkin's disease and tonsil sepsis, but lymphatic leukemia, blood dyscrasia, carcinoma of the mouth and pharynx, syphilis and tuberculosis. "What is the cause of pain in the ear?" They are supposed to show that it may be associated with a red drum or white drum, the former with a local lesion and the latter with a remote (referred) cause. A patient comes in with nosebleed. What happens? They are supposed, after they have stopped the nosebleed, to treat the man from the medical standpoint. We try to teach that otolaryngology is not a separate entity, but is a little adjunct to the practice of medicine, because these men go out into the country where they have nobody to whom they may refer patients. If a man is surgically inclined we try to make him very conservative in his ideas; we lay stress on the importance of medical treatment of ear, nose and throat patients.

Dr. Frank R. Spencer, Boulder, Colo.: Dr. Joseph C. Beck has asked me to tell you something about the investigation Dr. Burt R. Shurly and I made of graduate teaching of otolaryngology in America, a few years ago, at the request of the American Board of Otolaryngology. We found relatively few schools that do graduate teaching of otolaryngology were qualified to give complete courses. It seems to me that one of the most important functions of this teaching section will be to encourage those who are doing good work in graduate teaching of otolaryngology to continue and to encourage other schools to undertake the work on a thorough basis, because relatively few undertake it.

I am heartily in favor of Dr. Wherry's report.

INSTRUCTIONAL SUBJECTS

DR. JAMES A. BABBITT, Philadelphia, Pa.
Tinnitus Aurium

DR. GEORGE M. COATES, Philadelphia, Pa.
Conservative Surgery of the Middle Ear and Mastoid

DR. A. C. FURSTENBERG, Ann Arbor, Mich.
Osteomyelitis of the Skull

DR. C. J. IMPERATORI, New York, N. Y.
Cancer of the Larynx—Diagnosis and Treatment

DR. ED L. LINGEMAN, Indianapolis, Ind.
Surgical Treatment of the Acute Mastoid

DR. J. B. McMURRAY, Washington, Pa.
Evaluation of Nose and Throat Conditions as They Affect the Eye

DR. FRED J. PRATT, Minneapolis, Minn.
Indications for Tonsillectomy

DR. ARTHUR W. PROETZ, St. Louis, Mo.
Allergy

DR. ARTHUR W. PROETZ, St. Louis, Mo.
Sinus Diagnosis

DR. D. C. SMYTH, Boston, Mass.
Bronchoscopy as an Aid in the Diagnosis and Treatment of Diseases of the Lung

DR. FRANK R. SPENCER, Boulder, Colo.
Laryngeal Tuberculosis

DR. FRANK R. SPENCER, Boulder, Colo.
Surgical Anatomy of the Sinuses

DR. H. H. VAIL, Cincinnati, Ohio
Vidian Neuralgia—Anatomical, Diagnostic, Clinical, Pathological and Therapeutic Consideration

DR. W. P. EAGLETON, Newark, N. J.
Meningitis

DR. RALPH A. FENTON, Portland, Ore.
Immunity in the Field of Otolaryngology

DR. PERRY G. GOLDSMITH, Toronto, Canada
Traps and Pitfalls in the Practice of Otolaryngology

DR. L. K. GUGGENHEIM, St. Louis, Mo.
Embryology of the Ear, Nose and Throat

DR. L. M. HURD, New York, N. Y.
Indications and use of Diathermy in Otolaryngology

DR. S. IGLAUER, Cincinnati, Ohio
Peritonsillar, Retropharyngeal, Parapharyngeal, Deep Cervical Abscess, Post-tonsillitis Pyemia and Ludwig's Angina

DR. DE FOREST C. JARVIS, Barre, Vt.
Diet in Otolaryngology

DR. ED KING, Cincinnati, Ohio
Rhinological Aspect of Tear Sac Surgery

DR. HARRY W. LYMAN, St. Louis, Mo.
Diagnosis of Internal Ear Lesions

DR. C. F. McCLINTIC, Detroit, Mich.
Headaches

DR. C. F. McCLINTIC, Detroit, Mich.
Anatomy of the Base of the Brain

DR. W. MITHOEFER, Cincinnati, Ohio
Defects of the Singing and Speaking Voice and Their Treatment

DR. HARRIS P. MOSHER, Boston, Mass.
Present Day Standing of Major Diseases of the Esophagus

DR. S. J. PEARLMAN, Chicago, Ill.
Differential Diagnosis of Deafness

DR. FRED J. PRATT, Minneapolis, Minn.
Indications for Tonsillectomy

DR. ROBERT F. RIDPATH, Philadelphia, Pa.
Surgery of the Nasal Septum

DR. GEORGE L. TOBEY, Jr., Boston, Mass.
Acute and Chronic Otitis Media

DR. GABRIEL TUCKER, Philadelphia, Pa.
Types of Laryngeal Obstruction and Treatment

DR. THOMAS D. ALLEN, Chicago, Ill.
Anatomy and Physiology of Sympathetic Nervous System of the Eye

DR. S. J. BEACH, Portland, Maine
Refinements of Refraction

DR. NELSON MILES BLACK, Miami, Fla.
Injuries to the Lens and Their Management

DR. ALBERT L. BROWN, Cincinnati, Ohio
Physio-Therapy in Ophthalmology

DR. MARTIN COHEN, New York, N. Y.
Phlyctenular Kerato-Conjunctivitis

DR. ALFRED COWAN, Philadelphia, Pa.
The Schematic Eye of Gullstrand

DR. WILLIAM H. CRISP, Denver, Colo.
Demonstration of Use of Cross Cylinders

DR. SANFORD R. GIFFORD, Chicago, Ill.
Chemo-Therapy in Ophthalmology

DR. D. F. GILLETTE, Syracuse, N. Y.
Normal and Abnormal Pupillary Reactions

DRS. HARRY S. GRADLE AND PETER KRONFELD, Chicago, Ill.
Diagnosis of Simple Glaucoma

DR. W. B. LANCASTER, Boston, Mass.
Theory and Practice of Modern Illumination

DR. EDWARD JACKSON, Denver, Colo.
Changes in Refraction of Eyes with Age

DR. R. I. LLOYD, Brooklyn, N. Y.
Physiology of Binocular Vision and Its Practical Application to Squint and Routine Examination

DR. DONALD J. LYLE, Cincinnati, Ohio
Graphic Demonstration of Cerebral Syndromes

DR. C. S. O'BRIEN, Iowa City, Iowa
Diagnosis and Management of Secondary Glaucoma

DR. C. W. RUTHERFORD, Iowa City, Iowa
Technique of Perimetry

DR. M. S. SCHOENBURG, New York, N. Y.
Retinal Detachment and Its Surgery

DR. EDMUND B. SPAETH, Philadelphia, Pa.
Use of the Free Skin Graft in Plastic Surgery About the Eye

DR. ROBERT VON DER HEYDT, Chicago, Ill.
Use and Application of Contact Glasses

DR. WEBB W. WEEKS, New York, N. Y.
History Taking and Evaluation of Symptoms

DR. J. M. WHEELER, New York, N. Y.
Iridectomies of Various Types

DR. CHARLES A. BAHN, New Orleans, La.
Prophylaxis and Treatment of Gonorrheal Conjunctivitis

DRS. ARTHUR J. BEDELL AND H. P. WAGENER, Albany, N. Y. Rochester, Minn.
Projection and Discussion of Stereo-Photographs of the Fundis Oculi

DR. W. L. BENEDICT, Rochester, Minn.
The Choice of Operation in Cataract Extraction

DR. WILLIAM E. BRUNER, Cleveland, Ohio
Relation of Gastro-Intestinal Disease to Ocular Disease

DR. BEULAH CUSHMAN, Chicago, Ill.
Hereditary Malformations of the Eye

DR. W. C. FINNOFF, Denver, Colo.
Retinal Tuberculosis

DR. CLARENCE KING, Cincinnati, Ohio
Management of Chronic Iritis

DR. LUTHER C. PETER, Philadelphia, Pa.
Central Visual Field Defects

DR. H. H. TURNER, Pittsburgh, Pa.
Interstitial Keratitis—Diagnosis and Management

DR. MEYER WIENER, St. Louis, Mo.
Surgery of the Tear Sac

PRESENTATION OF INSTRUMENTS AND APPLIANCES

A CROSSED-WIRE MARKER FOR LOCALIZING THE TEAR IN DETACHMENT OF THE RETINA.

CONRAD BERENS, M. D.,

NEW YORK.

The crossed-wire marker, employed in localizing the tear in detachment of the retina, consists of a convex beveled ring, the diameter of which is 12.5 mm. The rigid metal of the ring is 1 mm. wide and has four small perforations, 90 degrees apart. Two

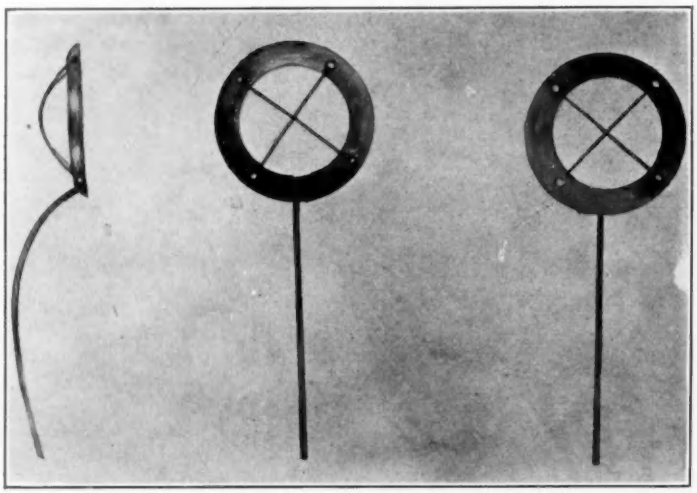

Figure 1. Crossed-wire marker for localization of tear.

pieces of thin wire, the radii of which are 5 mm., are extended from opposite perforations, crossing in the center. (See Fig. 1.) A handle is attached to the ring midway between two of the perforations.

TECHNIC OF USING THE CROSSED-WIRE MARKER.

After complete dilatation of the iris has been obtained with mydriatics and adrenalin, and one or two drops of a solution of

1 per cent holocain have been instilled, the crossed-wire marker is placed exactly over the apex of the cornea and the tear located by means of the ophthalmoscope. One of the crossed wires is adjusted to bisect the tear (See Fig. 2), the surgeon being certain that the point at which the wires cross corresponds to the apex of the cornea. A needle, previously dipped in a 1 per cent alcoholic solution of gentian violet, is used to prick the conjunctiva through the holes at the ends of the wire bisecting the tear. An assistant holds a black thread tightly stretched over these two points, and if this bisects the tear when viewed ophthalmoscopically, and repeated observations indicate that the marks are

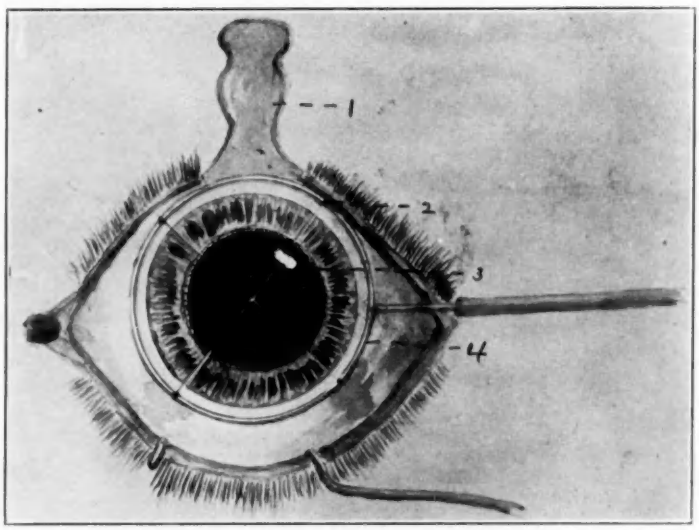

Figure 2. Crossed-wire marker in place for localization of tear.

1. Lid retractor.
2. Opening in ring of crossed-wire marker.
3. Iris margin.
4. Curved margin of crossed-wire marker.

in the correct position, a 26 gauge needle may be dipped in India ink and passed subconjunctively through the gentian violet spots for permanent marking. The location of the tear is again verified on the operating table by stretching a thread over the India ink marks and observing the tear with the ophthalmoscope. A black silk thread, knotted at one end, is passed toward the tear, first through the more distant ink mark and then through the ink

mark near the tear. This thread should pass over the apex of the cornea and bisect the tear. If this last thread is in exact position, it is extended over the exposed sclera and the point along this line where the tear has been located is marked with gentian violet. Ignipuncture may then be performed.

A CROSS ACTION IRIS FORCEPS.

SANFORD GIFFORD, M. D.,

CHICAGO.

This instrument was intended to make the maneuver of iridectomy simpler. With the usual forceps the iris must be grasped with the left hand and the forceps held shut, while it is withdrawn, and cut off with the right hand. Some operators develop a slight tremor during this procedure, which is otherwise never present. The cross-action forceps are introduced closed, opened slightly to engage the iris and allowed to close. The grasp is relaxed completely and the forceps withdrawn while held loosely between

Cross action iris forceps.

thumb and forefinger. The blades are curved less than the usual iris forceps, which allows their introduction while the hand rests in a natural position on the forehead. The blades are very delicate, but the handle is long so as to rest like a pen in the hand. The idea of a cross-action forceps was suggested by Mr. Arthur Sinclair's cross-action capsule forceps. Another advantage of the forceps is that it is impossible to let go of the iris involuntarily, while if it is necessary to do so a slight pressure will release it. Also since the blades are very fine and separated by positive pressure they will grasp the iris through a very small incision, which is of advantage in iridencleisis.

A BINOCULATOR.

NELSON M. BLACK, M. D.,

MIAMI, FLORIDA.

The binoculator is the name given by its originator, an optometrist of Fort Pierce, Florida, to the apparatus, by means of which graduated, uniform exercise may be given the extrinsic ocular muscles. It is made up of two balanced prisms made to

Binoculator.

rotate at a uniform speed, activated by a motor and gears. A fixation target placed before the prisms appears to rotate six or seven times clockwise, and the rotation is instantly changed to counter-clockwise. The amplitude of the apparent rotation of the

fixation target depends on the distance it is from the eyes. The eyes following the fixation point naturally have to rotate, thus every extrinsic ocular muscle is given even and uniform exercise.

By interposing prisms base out between the rotating prisms and target, additional effort is required of the adductors to prevent diplopia with the idea of increasing their strength. The same applies to the abductors with prisms base in, and to the vertical and oblique muscles so placed to add to their work.

For those who believe in orthoptic exercises for so much insufficiency, and I do, I feel there is a large field for use for an instrument of this character—to help build up divergence and convergence power, and in addition in certain pathologic intraocular conditions the exercise should be helpful, as it must increase the nutrition to the parts.

A BINOCULAR MAGNIFIER OR LOUPE.

NELSON M. BLACK, M. D.,

MIAMI, FLORIDA.

Having been annoyed and disgusted with the various loupes such as the Beebe and Zeiss, because of their bulk, the limited field of vision, and the projecting parts, eight years ago I attempted to overcome these objections and succeeded in doing so to my own satisfaction with this simple affair. The basic lens is made up of my presbyopic correction. The segments are convex lenses to give the magnification necessary, plus prisms base in, sufficient to do away with any effort of convergence on the user's part. In my case + 3.00 D. sph. combined with 5 degree prism base in, before each eye is necessary. The zone of presbyopic correction about the segment gives one the necessary unobstructed range of vision for reaching for treatment, etc. I am informed that some of the manufacturers of bifocals can make them up in one-piece lenses.

A SCLERAL TACK.

T. D. ALLEN, M. D.,

CHICAGO.

We all make mistakes in choosing the exact site on which we want to make a trephine opening in detached retina, and because of that I decided a small tack might be placed to one side

of the trephine hole before we use diathermy or the actual cautery. The tack is on the end of a small plate which has the curvature of the sclera. It is blunt, so if the trephine hole is made in the place you think the hole in the retina is, after you have gone through the choroid in order to evacuate the subretinal fluid you may put this tack in, and because it is blunt it will not make another hole in the retina in case you are not in the proper place. Because it is so thin and of the same curvature as the sclera, you can leave it in, although you have gotten almost back to the posterior fold of the eye, and then allow the eye to go back and with the ophthalmoscope look through the cornea into the eye and see if you can see the end of the tack, or if you are fortunate enough to have passed through the hole in the retina.

A PHOTOKERATOSCOPE.

ROBERT VON DER HEYDT, M. D.,

CHICAGO.

A photokeratoscope—photographing the corneal surface contour.

The old Placido disc image gives a picture of the corneal surface to the observer. Documentary evidence of this picture can best be obtained by photography. This latter subject has been studied by Gullstrand, Fischer and more recently by Dekking and Amsler. The Dekking apparatus, shown in 1929, is so constructed that it includes the whole corneal surface. The apparatus more recently constructed by Carl Zeiss for Amsler of Lausanne is simpler in that it depicts only the image projected onto the important central corneal area.

This photokeratoscope was first demonstrated at the Heidelburg Congress in 1930. It is useful in recording the images in regular and irregular astigmatism, corneal ulcers, scar contraction, loss of corneal substance and keratoconus. The images obtained by photography are very distinct and large enough to record clearly all minute changes in surface form and contour.

I have the privilege of demonstrating some of the pictures made with the Amsler apparatus.

APPARATUS FOR TESTING NEAR VISION.

F. Park Lewis, M. D.,

Buffalo.

It consists of lines of the same diameter on a movable slide. For example, by turning the wheel we get the largest lines, and by sliding the corresponding slide we get lines of equal amplitude. The dial has horizontal lines which when adjusted are in perfect alignment. For those who are illiterate and unable to read our letters, we find this a very valuable test. The finest line corresponds to about a one-point letter, and the largest to about a ten-point, and by turning the entire apparatus on the wheel these lines determine whether there is astigmatism. The whole thing is simple in application.

A RED-GREEN AUTOMOBILE GLASS.

V. A. Chapman, M. D.,

Milwaukee.

A red-green automobile glass for the color-blind driver.

This is a real problem in some states. The principle is simple. This is a pair of old spectacles—ordinary glass; and fastened on the top is a red-green filter. In driving, the man wears them the same as his other glasses. When he approaches a light and sees it brown or gray and does not know whether it is red or green, by dropping his head and looking through the upper part, if it is green it stays just the same, and if it is red it is perfectly white. A red-green color-blind driver with a pair of these glasses is just as safe as a man who is not color blind.

A HAND SCHEMATIC EYE.

H. H. Glosser, M. D.,

Buffalo.

This is used for the purpose of teaching. Suppose a doctor has a patient and desires to show the changes in the fundus, such as hemorrhage. Place the proper picture here, close the apparatus, turn on the light and then present the students with the exact picture in the patient.

MINUTES

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

1931

JOINT SESSION

MONDAY MORNING

The thirty-sixth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology was held at the French Lick Springs Hotel, French Lick, Indiana, September 13th to 19th, 1931.

The Monday morning session was called to order at nine o'clock by a Senior Past President, Dr. Edward Jackson of Denver.

DR. EDWARD JACKSON: The thirty-sixth Annual Meeting of the Academy will please come to order. Our method of opening today is an innovation. I do not know who suggested it or who is responsible for it. It came to me through the Secretary of the Academy, and his broad shoulders are capable of bearing the responsibility. But it occurs to me that it is a very good method of opening our meeting. Every step forward is from the preceding step. On such occasions we ought to have clearly in mind the past, out of which our life has come, and the possibility of what we can add to that life in the future. So I am very willing to open this innovation by simply introducing to you the President of the Academy, Dr. John F. Barnhill, who will welcome us to the State, and will give us an address that ought to draw a good audience before he is through. He is willing to live up to the letter of the program. It is mere form to introduce Doctor Barnhill, but I am very glad to use that form.

DR. JOHN F. BARNHILL: It is the habit of this Academy to begin on time. I think it is one of the great things in life—to begin on time, and while we are waiting for the members to

gather I would like to repeat the story Samuel D. Grose tells about being on time. Once upon a time, visiting a great internist in Paris, the internist said, "Come and see my work tomorrow at thirteen minutes after one." Grose said that thirteen minutes struck him, since in America we say fifteen or twenty minutes; but he was there the next day at twelve minutes past one—one minute ahead of time, and as he got out of his carriage in front of the hospital he saw this internist going up the steps—also one minute ahead of time. I thought that a very good story of a great internist who was one minute ahead of his time.

At this point Dr. Barnhill delivered his President's address. (See page 15.)

THE PRESIDENT: I now have one of the pleasantest duties of my life, and I daresay it is to this Academy one of its happiest moments—the introduction of our guest of honor. It is a curious situation because it has never happened but once before. It is a situation in which were I to waste any words I would probably be doing the occasion a serious wrong, but I do think I should stop long enough to say that when I, following the prerogative of the President, came to select the guest of honor, I found in America so much of the good in otolaryngology, and so many men who possessed that good, that I thought we should not go to a foreign country to select this guest. I did talk with a good many representative men as to whom I should appoint, and I wish to say this, that almost with one voice there came back to me the request, which met with my most hearty approval, to appoint the gentleman who has been appointed.

I feel it is a very great honor, indeed, to bring forward and introduce to you at this moment a man who has been an outstanding entity in otolaryngology for more than a quarter of a century. It is our own Joe Beck. You will now hear from our honor guest, Dr. Joseph C. Beck of Chicago.

DR. JOSEPH C. BECK: Mr. President, Ladies and Gentlemen —I have not the words to express my feelings. I would have to resort to the mother tongue I used almost fifty years ago when I came to this country—I have not the words to express in the English language what I feel in my heart for the honor that has been bestowed upon me by the Academy. The Academy has been everything to me in development, has given me the opportunity to do—nothing original, but to try to carry on the work of others,

and to try to tell the truth, absolutely and without reservation, as to the results. I appreciate most sincerely, Mr. President, your selecting me as the guest of honor.

THE PRESIDENT: We will now begin our scientific program with a symposium on "Malignant Diseases," the first paper by our guest of honor, Dr. Beck:

MALIGNANT DISEASES: NOSE—NASOPHARYNX—ORBIT.

Dr. Joseph C. Beck, Chicago.

Dr. Gordon B. New, Rochester, Minn.

Dr. Bernard Samuels, New York.

This symposium was discussed by Drs. W. L. Benedict, Rochester, Minn.; French K. Hansel, St. Louis, Mo.; Edward Jackson, Denver; John F. Barnhill, Indianapolis, Joseph C. Beck and Bernard Samuels.

The Monday morning session adjourned.

SECTION ON OPHTHALMOLOGY

TUESDAY AFTERNOON SESSION

The Tuesday afternoon session convened at one-forty-five, Dr. Walter B. Lancaster of Boston presiding.

Dr. Edward Jackson, Denver, Colo., read a paper entitled "Share of Crystalline Lens in Ocular Refraction." Discussed by Dr. Alfred Cowan, Philadelphia, and Dr. Edward Jackson.

Dr. C. S. O'Brien, Iowa City, Iowa, read a paper entitled "Astigmatism—Its Accurate Determination and Correction." Discussed by Drs. C. P. Clark, Indianapolis, Ind.; Louis Bothman, Chicago; Edward N. Robertson, Concordia, Kans.

Dr. Leo L. Mayer, Chicago, read a paper entitled "An Experimental Study of Detachment of the Retina and Its Surgical Therapy." Discussed by Drs. A. L. Brown, Cincinnati, O.; Sanford R. Gifford, Chicago, and Dr. Leo L. Mayer.

Dr. Walter S. Atkinson, Watertown, N. Y., read a paper entitled: "Angiomatosis Retinæ with Cerebellar Tumor." Discussed by Drs. A. J. Bedell, Albany, N. Y.; George F. Suker, Chicago, and Walter S. Atkinson.

The Tuesday afternoon session adjourned.

Tuesday Evening Session

Symposium on Aviation Medicine

The Tuesday evening session convened at eight-twenty, the President, Dr. John F. Barnhill, presiding.

The President: To many people who are interested in aviation it has been the belief that it may occupy a considerable part of the attention of the otolaryngologists in the future. When the war was on it was said that we could win the war with food; but when the Government said we should fill the skies with aeroplanes, then the otolaryngologists and ophthalmologists, the men skilled in knowing just what sort of man ought to fly, hand in hand with the aviator, the mechanic and others, began to make aviation safer than it would otherwise have been.

We are exceedingly fortunate tonight in having a symposium which covers subjects of intense interest to everyone who wishes in the future to live among the clouds.

The first paper on the program is on the "Government Aspect of Aviation," by Dr. Louis H. Bauer. Unfortunately, Dr. Bauer cannot be here, but I understand Dr. Nelson Black will read his paper.

The following symposium on Aviation was presented:

"Government Aspect of Aviation," Dr. Louis H. Bauer.

"Present Ophthalmological Standards for Commercial Aviation," Dr. Conrad H. Berens and Dr. Harry T. Smith.

"Present Otolaryngological Standards for Commercial Aviation," Dr. Ralph A. Fenton.

"The Present Status of Aviation in the United States," Hon. David S. Ingalls, Assistant Secretary, U. S. Navy.

The Tuesday evening session adjourned.

SECTION ON OTOLARYNGOLOGY

Wednesday Afternoon Session

The Wednesday afternoon session convened at one-forty, the President, Dr. John F. Barnhill, presiding.

Dr. Lyman G. Richards, Boston, read a paper entitled "The Prognostic Significance of Sinusitis in Children." Discussed by Drs. Arthur W. Proetz, St. Louis; John J. Shea, Memphis, Tenn.; A. M. Alden, St. Louis; M. F. Arbuckle, St. Louis; R. G. Means, Columbus, Ohio, and Lyman G. Richards.

THE PRESIDENT: We have the very great honor of having one of our members elected to the presidency of the great American Medical Association. It is an honor that has come to us but three times, and I wish to express your appreciation to Dr. Cary, who is present at this time.

DR. E. H. CARY: Mr. President, Ladies and Gentlemen—It is a very great honor to be a member of the Academy. The honor which has come to me is almost overwhelming, and it remains to be seen whether I shall measure up to its responsibilities. I will not detain you in the midst of this wonderful program. To me this Academy is one of the great teaching organizations of the country. I think postgraduate medical education has been emphasized and carried out and become more popular from the standpoint of the medical society than any other source. It is a great pleasure to me to come to the Academy each year and sit at the feet of these wonderfully equipped, beautifully trained men who can so well carry the message that means so much to the science of medicine.

It is a great honor to be here, Mr. President, and to be introduced by such a distinguished gentleman as yourself.

Dr. E. Fulton Risdon, Toronto, Canada, read a paper entitled "The Management of the Fractured Nose." Discussed by Dr. T. E. Carmody, Denver, Colo., and Dr. E. Fulton Risdon.

Dr. John J. Shea, Memphis, Tenn., read a paper entitled "Nasal Obstruction and Systemic Consequences." Discussed by Drs. G. F. Harkness, Davenport, Iowa; John A. Pratt, Minneapolis, Minn.; A. M. Alden, St. Louis; Frederick E. Hasty, Nashville, Tenn.; H. H. Vail, Cincinnati, Ohio, and Dr. John F. Barnhill, Indianapolis.

Dr. W. A. Wagner, New Orleans, read a paper entitled "Diagnosis and Conservative Treatment of Sphenoid Suppuration." Discussed by Drs. R. M. Armstrong, Lexington, Ky., and George B. Collier, New Orleans.

The Wednesday afternoon session adjourned.

SECTION ON OPHTHALMOLOGY.

THURSDAY AFTERNOON SESSION

The Thursday afternoon session convened at one-thirty, Dr. Meyer Wiener, First Vice-President, presiding.

Presentation of Instruments

(See page 381.)

Dr. S. B. Marlow, Syracuse, N. Y., read a paper entitled "Visual Fields in Chronic Glaucoma—The Effect of Reduced Illumination." Discussed by Drs. L. C. Peter, Philadelphia; M. J. Schoenberg, New York; and Dr. S. B. Marlow.

Dr. G. R. Callender, Washington, D. C., read a paper entitled "Malignant Melanotic Tumors of the Eye—A Study of Histological Types in 111 Cases." Discussed by Drs. J. S. Friedenwald, Baltimore; V. A. Chapman, Milwaukee; Martin Cohen, New York City; and Dr. G. R. Callender.

The Chairman thanked Major Callender for his courtesy in making this presentation.

Dr. F. Park Lewis, Buffalo, read a paper entitled "Cataract in Fishes." Discussed by Drs. Harvey Howard, St. Louis, and F. Park Lewis.

Dr. Alfred Cowan (in conjunction with Dr. W. E. Fry), Philadelphia, presented a paper entitled "The Hyaloid Membrane of the Vitreous." Discussed by Drs. R. C. Gamble, Chicago; Daniel B. Kirby, New York City; Robert von der Heydt, Chicago; V. A. Chapman, Milwaukee; M. J. Schoenberg, New York City, and Alfred Cowan.

The Thursday afternoon session adjourned.

TEACHERS' SECTION

Thursday Afternoon

The Teachers' Section convened Thursday afternoon at four-fifteen, the President, Dr. John F. Barnhill, presiding.

Dr. W. P. Wherry, Omaha, read a paper entitled "Purpose of Teachers' Section."

Dr. Duncan MacPherson, New York City (by invitation), read a paper entitled "Discussion of Problems in Graduate Teaching."

Dr. S. J. Kopetzky, New York City, presented the subject "Standardizing Graduate Courses in New York City by the New York Academy of Medicine."

The above papers were discussed by Drs. Burt R. Shurly, Detroit; Joseph C. Beck, Chicago; Martin Cohen, New York City; Perry G. Goldsmith, Toronto, Canada, and Frank R. Spencer, Boulder, Colo.

Moved by Dr. Harris P. Mosher that the plan outlined in Dr. Wherry's paper be adopted. Motion seconded and carried.

The Teachers' Section adjourned.

BUSINESS MEETING

The annual Business Meeting convened at eight o'clock, Thursday evening, September 17th, the President, Dr. John F. Barnhill, presiding.

The following reports were read:

Report of Executive Secretary-Treasurer

1931

First I wish to express sincere appreciation for the very fine work accomplished and the loyalty expressed in executing all proposed activities by the associated secretaries, the officers and others assisting in the general maintenance of the Academy.

The general organization is rapidly becoming a proper and smooth-running machine, to the end that the large mass of detail is now being handled without friction.

Membership Summary

Membership reported as of October 1, 1930:

In good standing	1,676	
Elected to membership at Chicago meeting	154	
Total, January 1, 1931	1,830	
During the year death has claimed		20

Dr. Hubert Arrowsmith, Brooklyn, N. Y.
Dr. George William Boot, Evanston, Ill.
Dr. Elmer T. Boyd, Denver, Colo.
Dr. John A. L. Bradfield, LaCrosse, Wis.
Dr. H. H. Briggs, Asheville, N. C.
Dr. George McClellan Burroughs, Danielson, Conn.
Dr. Wesley Lyman Curtis, Lincoln, Neb.
Dr. David N. Dennis, Erie, Pa.
Dr. Lawrence W. Fox, Philadelphia
Dr. Robert Clyde Lynch, New Orleans

Dr. Murray McFarlane, Toronto, Canada
Dr. R. H. T. Mann, Texarkana, Ark.
Dr. John W. Park, Harrisburg, Pa.
Dr. Joseph A. Stucky, Lexington, Ky.
Dr. M. M. Savage, Baltimore
Dr. James C. Tucker, Beatrice, Neb.
Dr. Derrick T. Vail, Sr., Cincinnati, Ohio
Dr. Ernest Cattron Wheeler, Tacoma, Wash.
Dr. Hiram Woods, Baltimore
Dr. James Woodson, Temple, Texas

The following Fellows have resigned	6

Dr. H. B. Graham, San Francisco
Dr. J. Guttman, New York City
Dr. William Howe Merrill, Lawrence, Mass.
Dr. J. Walter Park, Harrisburg, Pa.
Dr. Jesse B. Porter, Elkhart, Ind.
Dr. J. W. Croskey, Philadelphia

The following Fellows have been dropped for non-payment of dues	13

Dr. Seldon Cohn, Fulton, Ky.
Dr. J. P. DeRiver, San Francisco
Dr. P. J. H. Farrell, Chicago
Dr. George Gill, Elyria, Ohio
Dr. G. E. Gwinn, San Antonio, Texas
Dr. George A. Landes, Pampa, Texas
Dr. Joseph A. Lea, Fort Worth, Texas
Dr. E. E. Maxey, Aberdeen, Wash.
Dr. E. B. Miller, Philadelphia
Dr. Herman B. Miller, St. Louis
Dr. L. A. Newton, Oklahoma City
Dr. F. H. Roost, Sioux City, Iowa
Dr. Eugene Van Meter, St. Louis

	39
Total membership January 1, 1931	1,830
Membership carried forward	1,791
Candidates pending	177
Total carried forward on books as of September 1, 1931	1,968

FINANCE

The Academy has been operating during this current year under the new finance plan, and has been functioning very satisfactorily.

Two years ago, due to a finance reorganization, a $5,000.00 item was moved from our capital holding by the auditor. This has been satisfactorily adjusted and the item replaced with a bond for $5,000.00 and an additional 50 shares of common stock. Back interest, in part, was also adjusted. The Comptroller, Dr. Large, handled this item and deserves sincere commendation.

The books were audited as of September 1st by a Public Accountant and show

Capital holdings of	$102,227.72
Cash on hand	2,419.72
Total	$104,647.44

Expenses incurred for this convention will have to be paid from this reserve.

The Academy so far has been able to operate upon the $10.00 dues plan, which is very much less than any other national society of similar size and importance.

The Program Committee, as well as the Council, is sincerely interested to know the reaction of the membership to the many innovations tried at this convention. Those of you who have not expressed your opinion upon the ballots will confer a favor by so doing at once.

Sincerely yours,
W. P. Wherry,
Executive Secretary and Treasurer.

Moved by Dr. F. R. Spencer that this report be accepted. Motion seconded by Dr. Harry Pollock and carried.

Report of the Editors of the Transactions

September 1, 1931.

To the Council of the American Academy of Ophthalmology and Otolaryngology:

The editors have the honor to submit the following report concerning the publication of the Transactions of the Academy and the Presession volume for 1930:

TRANSACTIONS

Number of pages, 654.
Number of copies printed, 1960.

EXPENSE:

Production—		
Typesetting, printing and binding	$3918.38	
Engraving	317.08	
Petty expense (printer's and Editors' offices, Transactions and Presession volume)	70.60	
	$4306.06	
Deduction authors' share in engraving expense	121.70	
		$4184.36
Distribution—		
Cartons, labels, zoning, wrapping and mailing 1765 copies	117.45	
Postage 1765 copies	405.30	
		$ 522.75
TOTAL EXPENSE		$4707.11

PRESESSION VOLUME

Number of pages, 320.
Number of copies printed, 1750.

EXPENSE:

Production—		
Printing and binding	$661.00	
Resetting for Transactions and type not used in Transactions	68.00	
Remake for Transactions	90.68	
		$819.68
Distribution—		
Covers, wrapping, zoning and mailing	51.40	
Postage 1700 copies	218.89	
		$270.29
TOTAL EXPENSE		$1080.97

One hundred twenty copies of the membership list, 116 pages and cover, were supplied by the printer gratis.

COSTS PER COPY

Transactions:	
Production (1960 copies)	$2.134
Distribution (1765 copies)	.297
Total	$2.431
Presession volume:	
Production (1750 copies)	$0.468
Distribution (1700 copies)	0.159
Total	$0.627

These costs, relative to the number of pages in the book, compare favorably with previous issues.

The delay in publication was the result of unforeseen circumstances, beginning with dilatory authors and ending with press trouble and the failure of a paper mill—circumstances unlikely to recur another year.

In view of the permanent nature of this record, the materials in the book were selected with great care, the paper having been especially chosen for durability and freedom from discoloration with age.

The Editors are keenly mindful of the problems arising in the publication of remarks made, often without reflection or preparation, from the floor, and invite the guidance of the Council in such matters, as well as in any others which may occur to them. They have purposely avoided any form of censorship, regarding this as the function of the Section Chairmen, presiding over the discussions. They respectfully request the authors of discussions to submit for publication only those remarks actually made from the floor within the time limit permitted by the chairman of the session.

A list of suggestions for the preparation of manuscripts and illustrations has been mailed in advance of the present meeting to all members appearing on the program, in an effort to reduce avoidable expense and, through uniformity of material, to improve the general appearance of the coming book.

Respectfully submitted,

ARTHUR W. PROETZ.
LAWRENCE POST.

REPORT OF THE NECROLOGIST
1931

The Academy has lost the following members by death since the last meeting:

Dr. Hubert Arrowsmith

Dr. Hubert Arrowsmith of Brooklyn died there on August 9, 1931, in his sixty-sixth year. A pioneer in bronchoscopy, Dr. Arrowsmith was a founder of the American Bronchoscopic Society. He had practiced medicine continuously in Brooklyn since he was graduated from the Long Island College Hospital, where he received his degree in 1886.

Dr. Arrowsmith was a Fellow of the American Laryngological Association and a member of the American Laryngological, Rhinological and Otological Society, the American Academy of Ophthalmology and Otolaryngology, the American Bronchoscopic Society, the American College of Surgeons and the American Medical Association.

About two years before his death Dr. Arrowsmith virtually retired from active practice. Prior to that time he was laryngologist to Kings County Hospital, St. Peter's Hospital, the Brooklyn State Hospital, the Jewish Hospital, the Brooklyn Hospital and St. John's Hospital, all of Brooklyn. He was also consulting laryngologist at the Huntington Hospital, Huntington, L. I., and St. Anthony's Hospital in Wood Haven, Queens.

He is survived by his widow, Mrs. Josephine Howard Arrowsmith, three daughters, the Misses Miriam and Eleanor Arrowsmith, and Mrs. John Keith Baker of 51 South Oxford street, Brooklyn, and by a sister, Miss Edith Arrowsmith.

Dr. William Ford Blake

Dr. William Ford Blake of San Francisco, California, died August 28, 1931, in Palo Alto, California, at the age of fifty-seven years.

Dr. Blake graduated from the Cooper Medical College, San Francisco, California, in 1903.

He was a member of the California Medical Association, the American Academy of Ophthalmology and Otolaryngology, and a Fellow of the American College of Surgeons.

Dr. George William Boot

Dr. George William Boot was born at Wilton, Iowa, on March 3, 1870, and died in Evanston, Illinois, June 21, 1931.

Dr. Boot was graduated from Sioux City Medical College in 1893 and from the University of Pennsylvania in 1898.

He was Chief of Staff of St. Francis Hospital, Evanston, and attending laryngologist and otologist at Cook County Hospital, Chicago. Previously he had been Assistant Professor of Otology at Rush Medical College, and Assistant Otologist at the Presbyterian Hospital.

During the last five years Dr. Boot had devoted himself to research work to find a possible cure for cancer. He was head of the Cancer Research Committee of St. Francis Hospital.

He was Captain, Medical Corps, U. S. A.

He was a member of the American College of Surgeons, American Medical Association, Chicago and Illinois State Medical Societies, American Academy of Ophthalmology and Otolaryngology, the Chicago Academy of Science, the Illinois Academy of Science, also the Chicago Laryngological and Otological Society.

Dr. Elmer Thomas Boyd

Dr. Elmer Thomas Boyd was born in Missouri, and died on November 24, 1930, at the age of sixty-six.

His medical career began when he became a pharmacist. Later he entered the Missouri Medical College to prepare for the practice of medicine. After graduation he returned to his home in Texas and began practice in Waxahachie. Desiring to prepare for special service he took the courses at the Manhattan Eye and Ear Infirmary.

In 1892, Dr. Boyd left Texas and went to Colorado, settling in Leadville. Subsequently he went to Denver and continued in his chosen field. In Leadville and in Denver he was oculist and aurist for the Colorado and Southern Railroad. Just before he passed away he was appointed to fill a similar position with the Denver and Salt Lake Railroad.

Dr. Boyd was a member of the Medical Society of the City and County of Denver, the Colorado State Medical Society. For many years he was a member of the Colorado Ophthalmology Society and for two years was its secretary. He was a member

also of the Academy of Ophthalmology and Otolaryngology, and the American Medical Association.

Dr. Boyd left surviving him his widow and one son.

Dr. John A. L. Bradfield

Dr. John A. L. Bradfield was born in East Fairfield, Ohio, in 1861, and died suddenly, while on a pleasure trip, on March 25, 1931, at Punta Gorda, Florida.

Dr. Bradfield attended Eureka College in Illinois, and, later, the College of Physicians and Surgeons in Chicago, graduating with the class of 1888. He was in general practice for two and one-half years at Loyal, Wisconsin, before specializing in eye, ear, nose and throat.

In 1890-1891 he attended the Chicago Polyclinic and Illinois Charitable Eye and Ear Infirmary. In the Fall of 1891 he located in LaCrosse, Wisconsin, limiting his practice to diseases of the eye, ear, nose and throat. During 1895 and 1896 he attended clinics in London and Vienna. He was a member of the regular staff and Advisory Board of St. Francis Hospital.

Dr. Bradfield organized the LaCrosse County Medical Society, which held its meetings in his office for several years. He was also a member of the State Medical Society, the American Medical Association, the American Academy of Ophthalmology and Otolaryngology, and was a Fellow of the American College of Surgeons.

His widow and two daughters survive.

Dr. Henry Harrison Briggs

Dr. Henry Harrison Briggs was born at Flag Pond, Tennessee, near the North Carolina line, on February 20, 1871, and died on February 2, 1931, aboard ship, while on a West Indian cruise in search of health.

His early education was received in the schools of his vicinity and at Tusculum College, Greenville, Tennessee. After graduating from this college he entered the academic department of Yale University and received his diploma from its medical school in 1897.

For several years he continued the study of his special line of work in New Haven and at the Manhattan Eye, Ear, Nose and Throat Hospital in New York City. In 1900 he came to Asheville, where he practiced until his death.

Dr. Briggs was a member of the following organizations: American Medical Association, American College of Surgeons, American Ophthalmological Society, American Academy of Ophthalmology and Otolaryngology, American Laryngological, Rhinological and Otological Association.

Dr. George McClellan Burroughs

Dr. George McClellan Burroughs was born at Sugar Grove, Pennsylvania, October 24, 1862, and died in Danielson, Connecticut, March 1, 1931. His wife and two daughters survive him.

Dr. Burroughs was graduated from Baltimore Medical College in 1900, and after postgraduate work abroad began practice in Danielson in 1904, giving especial attention to ophthalmology.

He was a member of the Killingly Medical Society, of which he was a founder, a past-President of the Windham County Medical Society, and a past Vice-President of the Connecticut State Medical Society. He was also a Fellow of the American Medical Association, of the American Academy of Ophthalmology and Otolaryngology, and of the American College of Surgeons.

Dr. Burroughs served the Day Kimball Hospital of Putnam, Connecticut, for many years as ophthalmologist and President of the Staff. He was also Medical Examiner of the town of Killingly.

His community activities included service as a trustee of the Brooklyn Savings Bank, and president of the Danielson Building and Loan Association.

During the latter years of his practice Dr. Burroughs was handicapped by ill health, but he never faltered in giving notably efficient and painstaking attention to his patients or in his attendance at all available society meetings. Born of a family of physicians, he had a reverence for medical ethics too seldom seen nowadays. He took almost a fatherly interest in every young physician coming in contact with him.

Dr. Wesley Lyman Curtis

Dr. Wesley Lyman Curtis was born at Tecumseh, Nebraska, January 7, 1870, and died in Omaha, Nebraska, on March 9, 1931.

Dr. Curtis was educated in the public schools of the State and at the Omaha Medical College. Upon receiving his medical degree

from Rush Medical College in 1896, he was admitted to practice at Omaha, Nebraska. He became an instructor in anatomy at the Omaha Medical College in 1897, and was on the teaching staff for six years, maintaining at the same time general practice in Omaha.

In 1905, Dr. Curtis went to the Phillipine Islands as surgeon of the Coast and Geodetic Survey, remaining on the islands two years, serving one year as resident surgeon of St. Paul's Hospital at Manila. Returning to Nebraska in 1908, he established an eye, ear, nose and throat practice at Fairbury, Nebraska. In 1918 he went to Lincoln, Nebraska, to practice his specialty with Dr. J. J. Hompes, which association continued until the date of his death.

Dr. Curtis was a member of the Nebraska Medical Association, and was President of the Lancaster County Medical Society in 1928. He was a Fellow of the American College of Surgeons and a member of the American Academy of Ophthalmology and Otolaryngology.

Dr. David Nichols Dennis

Dr. David Nichols Dennis, ophthalmologist, was born at Grafton, Massachusetts, December 23, 1858, and died at Erie, Pennsylvania, November 15, 1930.

He graduated at Jefferson Medical College in 1881, beginning his professional career in the hospitals of Philadelphia. Subsequently he practiced general medicine for a brief period at Killingly, Connecticut, and Worcester, Massachusetts, after which he became hospital assistant at the Jefferson Medical College in Philadelphia. In the latter institution he was assistant to Professor William Thompson, who was one of the early ophthalmologists in the United States. Dr. Dennis settled in Erie, Pennsylvania, in 1885, and at first specialized in diseases of the eye and ear, but later confined himself to diseases of the eye. He served as ophthalmic surgeon at the Hamot Hospital, Erie, Pennsylvania, and for a period of twenty years was consulting ophthalmologist to the Bessemer Railroad. He assisted in forming the Erie County Medical Society Library, and was twice president of the Society.

He was a Fellow of the American Medical Association, American College of Surgeons, American Academy of Ophthalmology and Otolaryngology, American Ophthalmological Society, the Buffalo (New York) Ophthalmologic Club, and a member of

the Pennsylvania Medical Society. At one time he was a director of the Erie Chamber of Commerce, which was later merged with the Board of Commerce.

For a number of years, until 1916, he was special examiner for the United States Pension Department, and was at one time director of the associated charities of Erie, and of the Social Service Federation, later called the "Community Chest."

Dr. Dennis published a number of articles dealing chiefly with the eye, ear, nose and throat, in various medical journals.

His widow and three children survive him.

Dr. Lawrence Webster Fox

Dr. Lawrence Webster Fox of Philadelphia, Pennsylvania, died June 4, 1931, at the age of seventy-eight years.

Dr. Fox was graduated from Jefferson Medical College of Philadelphia in 1878 and from the Medico-Chirurgical College of Philadelphia in 1896.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and the American College of Surgeons; Professor of Ophthalmology, Graduate School of Medicine of the University of Pennsylvania, and formerly Professor of Ophthalmology, Medico-Chirurgical College of Philadelphia and the University of Pennsylvania School of Medicine; Ophthalmologist to the Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases.

Dr. Robert Clyde Lynch

Dr. Robert Clyde Lynch was born in Carson City, Nevada, September 8, 1880, and died May 12, 1931, from injuries received in an automobile accident while driving with Dr. Joseph A. Stucky, who was instantly killed.

Although a Nevadan by birth, Dr. Lynch moved in early childhood to New Orleans, with which city he has been inseparably identified. He received both his preliminary and medical education there, graduating from Tulane Medical School in 1903. He occupied himself with general medicine for less than two years, and then began the study of otolaryngology in Tulane. After a year there he continued his studies abroad.

On his return, Dr. Lynch was associated with Dr. A. W. DeRoaldes, whom he succeeded as Surgeon-in-Charge of the New Orleans Eye, Ear, Nose and Throat Hospital, and at his

death was Professor of Otolaryngology in the Post-Graduate School of Medicine at Tulane University, and Consultant in Otolaryngology in the Touro Infirmary.

Dr. Lynch was a Fellow of the American Laryngological Association, the American Laryngological, Rhinological and Otological Society, the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the American College of Surgeons, and a past-President of the American Bronchoscopic Society. He was, from its inception, a member of the American Board of Otolaryngology. For many years he served on the Editorial Board of the Annals of Otology, Rhinology and Laryngology, and the Archives of Otolaryngology.

He was known especially for his successful development of the suspension method of laryngoscopy. In 1924 he described in detail a technique for an external radical pansinusitis operation, for which he claimed no originality, but which he described so meticulously that it is often called by his name. He had a special interest in photography and was one of the first to accomplish cinematography of the larynx. He was pre-eminently a teacher, and it was during a visit to a neighboring laryngological society, which he was to address, on May 12, 1931, that he met his untimely death, a tragedy not only for his immediate family, but for American laryngology.

He is survived by his widow and two sons.

Dr. Robert Howell Taylor Mann

Dr. Robert Howell Taylor Mann was born on September 24, 1868, at Brownsville, Tennessee, and died at Texarkana, Arkansas, April 17, 1931.

He received his medical education in the Vanderbilt University School of Medicine at Nashville, and graduated with the degree Doctor of Medicine in 1893. Following this he practiced with Dr. G. C. Savage of Nashville, receiving special training in eye, ear, nose and throat work. Dr. Mann then took post-graduate work in the Eye and Ear Hospital, and the Manhattan Eye and Ear Hospital, New York City, following which he located for the practice of medicine in Texarkana, Arkansas-Texas.

Dr. Mann was a pioneer in the reorganization of the State Medical Association, and had much to do with the successful career of the organization as it now stands. He had been a member continuously in good standing of the Bowie County Medical

Society, the State Medical Association, and the American Medical Association. Because of the location of his native city, he occupied a somewhat dual rôle in membership. He also belonged to the Arkansas State Medical Society, of which society he was President in 1928-29. He was a past-President of the Northeast Texas District Medical Society, and also served as President of the Tri-State Medical Society of Louisiana, Texas and Arkansas.

He was one of the founders of the Texarkana Hospital, joining with Doctors T. F. Kittrell, S. A. Collom and E. L. Beck, more than thirty years ago, in its establishment. He has since been a member of the Board and was its Secretary at the time of his death. His scientific attainments in his chosen specialty were of the highest type. He was a member of the American Academy of Ophthalmology and Otolaryngology.

He was one of the organizers of the levee system in Miller County, and remained active on the various boards of this system until the time of his death.

He is survived by his widow and three children.

Dr. John Murray McFarlane

Dr. John Murray McFarlane was born in Toronto, Canada, and died in that city on May 26, 1930, at the age of sixty years.

He received his education in Upper Canada College and Trinity Medical College, and took post-graduate work in Vienna.

Dr. McFarlane was a member of the Ontario Medical Association, the Royal Canadian Yacht Club, and other associations, including the American Academy of Ophthalmology and Otolaryngology.

He left surviving him his widow.

Dr. John Walter Park

Dr. John Walter Park was born at Carsonville, Pennsylvania, on October 22, 1855, and died at Harrisburg, Pennsylvania, on January 27, 1931.

He received his early education in the common schools. In 1872 he began the study of medicine with Dr. Caslow of Halifax, Pennsylvania. At the end of three years he entered Jefferson Medical College and graduated in 1873. He located in Williamstown, Pennsylvania, in 1879, where he engaged in general practice until 1889, when he went to Europe to take up the study of

the eye, ear, nose and throat. He was Clinical Assistant at the Royal London Ophthalmic Hospital, London, England, and assisted at the South London Eye Hospital. He then continued his studies in Berlin and Paris.

In 1889 he returned to New York and entered the New York Ophthalmic and Aural Institute, where he studied under Professor Knapp. In March, 1890, he located in Harrisburg, Pennsylvania, and was elected to the Harrisburg Hospital Staff the same year. He served on that staff until his retirement from hospital work in 1922.

Dr. Moses M. Savage

Dr. Moses M. Savage was born December 9, 1871.

He received his preliminary education at a German Gymnasium, took up pharmacy at the age of 16, passed his examination as Assistant Pharmacist at the Russian University of Dorpat. In 1892 he passed the Maryland Board of Pharmacy.

He graduated from the College of Physicians and Surgeons (University of Maryland) in 1895 and passed the State Board in the same year. In 1897 he was demonstrator of Clinical Medicine at his Alma Mater. In 1899 and 1900 he did post-graduate work at Johns Hopkins Hospital, including a course in Clinical Microscopy, and followed Dr. Osler's Clinics for several years. In 1907 and 1908 he was a demonstrator of Medical Topography, Physical Diagnosis and Laryngology at Baltimore Medical College, and from 1910 until his death was connected with the Nose and Throat Department of the Sinai Hospital, and in later years was a member of the staff.

He was physician to the Hebrew Benevolent Society and the Hebrew Friendly Inn and Aged Home for a number of years.

He was appointed Coroner of Baltimore City, serving during the years 1914 and 1915.

In 1921 he spent several months doing research work in the Clinics of Vienna. Upon his return he was appointed to the staff of the West Baltimore General Hospital and the Jewish Hospital for Consumptives.

He was a member of the Medical and Chirurgical Faculty, Southern Medical Association, American Academy of Ophthalmology and Otolaryngology, Fellow of the American Medical Association, and held the certificate of the American Board of Oto-Laryngology.

He died at Baltimore, June 25, 1928.

Dr. Joseph Addison Stucky

Dr. Joseph Addison Stucky was born in Louisville, Kentucky, September 6, 1857, and died on May 12, 1931, having been instantly killed while driving with Dr. R. C. Lynch of New Orleans on the Dixie Highway near Lexington, when their car collided with a motor bus, Dr. Lynch surviving only a few hours. The accident occurred as they were returning to Lexington after a visit to Berea College.

Dr. Stucky was best known, perhaps, for the establishment by him of clinics in the Kentucky mountains for the study and treatment of trachoma. From small beginnings, thirty years ago, these clinics have increased in number and size. He was instrumental in having located at Richmond a Government hospital for the treatment of trachoma. During the World War he served as consultant in the Medical Reserve Corps.

He was an active member of numerous national medical societies, and was former President of the American Academy of Ophthalmology and Otolaryngology, and the American Laryngological, Rhinological and Otological Society. He was a Fellow of the American College of Surgeons, and a member of the Lexington Medical Society, the Fayette County Medical Society, the Kentucky State Medical Society, the Mississippi Valley Medical Society, the Southern Medical Association, and the American Medical Association.

Dr. Stucky was the author of many articles which have been published in medical journals, and was a lecturer on public health and social welfare subjects. He was also a delegate to the International College of Surgeons in London, in 1912. For many years, and until his death, he was a member of the Editorial Board of the Annals of Otology, Rhinology and Laryngology.

Dr. James Cyrus Tucker

Dr. James Cyrus Tucker, aged forty-five, died at his home in Beatrice, Nebraska, Friday afternoon, July 3, 1931, of uremia.

He was graduated by the University of Nebraska College of Medicine in 1912. Following his graduation he practiced at Long Pine, Nebraska, until 1917, at which time he entered the Army as Captain in the Medical Corps. He served as Chief of the Ear, Nose and Throat Department at Base Hospital No. 1, in Paris, and during this time he was commissioned a Major.

Following his release from the military service Dr. Tucker spent one year in post-graduate study in the Post-Graduate School of the University of Pennsylvania. From 1920 to 1922 he was associated with Drs. Gifford, Patton, Callfas, Potts and Cassidy in Omaha, Nebraska. In 1922 he moved to Beatrice, Nebraska, where he continued to practice until the time of his death.

Dr. Tucker was a member of the American Academy of Ophthalmology and Otolaryngology, the American Laryngological, Rhinological and Otological Society, the Nebraska Eye and Eye and Ear Academy (President in 1927), and State and local societies.

Dr. E. C. Wheeler

Dr. E. C. Wheeler was born at Manchester, Iowa, on November 8, 1872, and died in Tacoma, Washington, 1931.

Dr. Wheeler was a graduate of the Manchester (Iowa) High School and was given a bachelor of arts degree at Cornell, later graduating from Rush Medical College at Chicago, in 1896. He did post-graduate work in Vienna and in London. He had been in active practice in Tacoma since 1903.

Dr. Wheeler, in September, 1917, entered the service of the United States and went immediately to France, where he served as a Major in the Medical Corps until after the Armistice.

He entered private practice again in April, 1919.

At the time of his death, Dr. Wheeler was a member of the Pierce County Medical Society, of the American College of Surgeons, of the American Academy of Ophthalmology and Otolaryngology, and had been given recognition as president of the Puget Sound College of Ophthalmology.

His widow and two children survive him.

Dr. Hiram Woods

Dr. Hiram Woods was born in Baltimore, Maryland, November 11, 1857, and died January 15, 1931. He graduated from Princeton University in 1879. In 1882 he graduated in medicine at the University of Maryland.

Since 1885 he confined his practice to Ophthalmology and Otology. He was demonstrator of anatomy at the University of Maryland from 1885 to 1888. From 1887 to 1894 he was Pro-

fessor of Ophthalmology and Otology at the Women's Medical College, Baltimore, now closed. From 1894 to 1920 he was lecturer and professor of Ophthalmology and Otology at the University of Maryland, and, in 1924, this university conferred upon him the honorary degree of LL.D.

He was surgeon to the Presbyterian Eye and Ear Hospital in Baltimore until 1917, and Ophthalmic Surgeon at the Union Protestant Infirmary, Hospital for the Women of Maryland. He was consultant at the Shepherd and Enoch Pratt Hospital and at the Washington County Hospital.

He was president of the Medical and Chirurgical Faculty of Maryland, in 1906, and chairman of its Council for several years; Chairman of the Section of Ophthalmology of the American Medical Association, 1912; President of the American Ophthalmological Society, 1919; and a member of the American Board of Ophthalmic Examinations from 1915 to 1920, and again from 1925 to 1926. He was a member of the Baltimore State Medical Society, Southern Medical Association, American Otological Society, American Academy of Ophthalmology and Otolaryngology, and a Fellow in the American College of Surgeons. For a number of years he was one of the directors of the National Society for the Prevention of Blindness and a very active member of the Maryland Society for the Prevention of Blindness. A meeting of the latter society at his office had been called for the day of his death.

He is survived by his widow and four children.

Dr. James Madison Woodson

Dr. James Madison Woodson was born January 2, 1868, and died September 30, 1930, at Temple, Texas.

Dr. Woodson was a graduate of Tulane School of Medicine, New Orleans, Louisiana, and of the Physicians and Surgeons' Medical College, New York City.

He was associated with the Scott and White Hospital since the beginning of his practice in Temple. He did the eye, ear, nose and throat work for this institution since its beginning. In 1923 he organized the Woodson Eye, Ear, Nose and Throat Hospital, and continued in close association with the Scott and

White Hospital. He, however, continued to do all of the eye, ear, nose and throat work for the Scott and White Hospital, acting as a department of the Scott and White Hospital and also being an independent organization. Dr. Woodson was engaged in the practice of eye, ear, nose and throat work in the Woodson Hospital with his two sons, Dr. Palmer Woodson and Dr. Burbank Woodson, at the time of his death. These two sons and one daughter survive him.

Respectfully submitted,

H. W. LYMAN, M. D.,
Necrologist.

This report was accepted and the members stood with bowed heads for one minute in memory of the departed.

REPORT OF AMERICAN BOARD OF OPHTHALMIC EXAMINATIONS

1931

The work of the Ophthalmic Board has been going on as usual in a very successful manner. We have held our two regular examinations this year, and one extra examination at Denver, at which time we examined nine candidates. It is the policy of this Board to hold examinations twice a year, at the meeting of the Academy and at the meeting of the American Medical Association; but we also occasionally, under justified conditions, hold examinations in some other part of the country. It is the policy to have no more extra examinations than necessary.

The standard of the men coming up seems to be improving each year; they are passing better examinations. And one of the interesting things about this Board is that there are a number of men who have been practicing for twelve or fifteen or more years who are coming up for certification. The Board sometimes requires simply case records under such conditions, if a man is vouched for by good men. Sometimes when we are not quite sure of their qualifications we give practical examination, but most of the men are not required to take written examination.

DR. LUTHER C. PETER.

REPORT OF AMERICAN BOARD FOR OTOLARYNGOLOGY

1931

The Board has held its two usual examinations, one in Philadelphia at the time of the American Medical Association;

one in Indianapolis, immediately preceding this meeting. Also an extra examination in Los Angeles. The following is a resumé of the Board's activities:

Los Angeles	61	
Philadelphia	79	
Indianapolis	34	174
Previously examined		1,718
Total certificated		1,892

Those who failed or were conditioned constitute about 12½ per cent.

DR. T. E. CARMODY.

REPORT OF COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY

1931

The Committee on Optics and Visual Physiology has held four meetings in the past year.

We are investigating visual acuity, the standardization of test charts and colored lenses, ophthalmic instruments and other subjects.

The committee requests $500.00 from the Academy for the further investigation of visual acuity and other subjects.

Respectfully submitted,

S. J. BEACH,
CONRAD BERENS,
WILLIAM BENEDICT,
E. V. L. BROWN,
E. C. ELLETT,
CHARLES A. BAHN,
WILLIAM WILDER,
EDWARD JACKSON,

Committee.

This report was referred to Council.

Moved by Dr. Burt R. Shurly that this report be adopted. Motion seconded and carried.

Report of Research Council in Pathology
1931

Nine years ago this Academy voted to establish a Section on Ophthalmology in the Army Medical Museum. You all know the work that has been done there in the past. To the original organization has been added members of the American Ophthalmological Society and of the Section on Ophthalmology of the American Medical Association. In the original scheme of things the pathology was supposed to have been both ophthalmic and otolaryngologic, but, as is rather common, otolaryngology lagged behind and it became the section on ophthalmic pathology—with all due respect.

The Academy has been responsible for this section and the large success it has attained. We have in the Museum nearly 50,000 specimens, of which nearly 2,000 are completely enucleated eyes, all of which have been studied and worked out in detail and the findings sent to the donor, with slides of what the eye showed.

It has become a heavy burden for the Army Medical Museum, a greater burden than the War Department would permit. Owing to the present money crisis the War Department has been cut down, and naturally the first part to suffer was the medical department. The Army Medical Museum staff was cut down and the increased work has become more than the Museum personnel could carry on. To the Museum has been added two tumors—lymphatic tumors and bladder tumors, and to carry on this work with that of the section on ophthalmology, the Research Council has volunteered to step in and provide one full-time pathologist and the requisite technician, thus relieving the Academy in the future of the financial contribution we have been making in the past. This was explained thoroughly to the Council and has received its approval, so that in the future the Section on Ophthalmology of the Army Medical Museum will be conducted exactly as in the past, subject to the same jurisdiction of the Academy as in the past, but without the financial support, therefore relieving the Academy of an appropriation of about $500.00 a year.

Dr. Harry S. Gradle.

Moved by Dr. T. E. Carmody that this report be accepted. Motion seconded and carried.

Army Medical Museum, War Department,
Washington, D. C., August 21, 1931.

The following is the annual report of the Division of Ophthalmic Pathology at the Army Medical Museum to the Committee on Pathology of the American Academy of Ophthalmology and Otolaryngology:

	Eye Specimens Received and Reported	Eye Specimens Diagnosed When Received	Other Items, Illustrations, Models, Etc.	TOTAL
For 7 yrs. Ending Dec. 31, 1927	800	583	1124	2484
For year Ending Dec. 31, 1928	177	46	——	223
For year Ending Dec. 31, 1929	305	21	0	326
For year Ending Dec. 31, 1930	275	202	5	477
Jan. 1, 1931, to July 1, 1931	215	0	0	215
Total,	1772	852	1129	3725

One hundred and eleven cases of malignant melanotic tumors are being followed annually and a report based on this group will be made to the Academy at the current meeting.

Forty-five cases of retinoblastoma had been received up to January 1, 1931. The age at enucleation is from less than 1 to over 10 years. The involvement was unilateral in 33 cases and bilateral in 12. The nerve was involved in 13 cases and in 17 more there was extension outside the globe. The duration before operation varied from one month to four years. Of the cases which have been followed to date, five are living, over five years after enucleation, three over 4 years, four over 3 years, four over 2 years, and two over a year. There were fourteen deaths reported from tumor, all occurring in less than a year after enucleation. There are no reports as to the outcome of six cases. Four of the fatal cases had extensions into the nerve beyond the lamina cribrosa, ten had extensions beyond the globe and nerve—that is, extensions into the orbital tissues. There have been reported, therefore, no deaths from the tumor when it had not extended beyond the limits of the globe itself.

The undersigned has not been informed officially of any action taken by the council with reference to the American Registry of

Pathology for the purpose of financing a continuation of this work. The table above indicates rather marked increase in the work and the necessity for such action. It is recommended that the Council of the American Academy of Ophthalmology and Oto-Laryngology give official approval to this effort and take whatever means may be at their disposal to assist in obtaining funds to place the whole project on a sound, permanent financial basis.

GEO. R. CALLENDER, Registrar.

REPORT OF COMMITTEE ON PUBLICITY AND LEGISLATION

1931

The publicity for the present meeting of the Academy was very good. The nose and throat journals were more favorable to the committee than the eye journals, the Annals, the Archives and the Laryngoscope giving us very favorable advance notices of the meeting. The Indianapolis people coöperated in every way with the committee, so we had very good publicity for this meeting.

DR. JOHN J. SHEA.

Moved by Dr. T. E. Carmody that this report be accepted. Motion seconded and carried.

REPORT OF COMMITTEE ON TUNING FORKS AND TESTS OF HEARING

1931

Last year your committee reported on sounding rods of the pitch of c-5 (4096 double vibrations) and c-6 (8192 double vibrations) which had been perfected by Mr. B. E. Eisenhour of the Riverbank Laboratory, Geneva, Illinois. These rods were made of the same aluminum-magnesium alloy of which the newest series of forks are constructed. Attention was called to the fact of the great advantage of the c-5 (4096 double vibrations) rod, which was that it vibrated from 50 to 90 seconds, whereas the very finest steel forks do not vibrate more than 20 to 30 seconds. It is, therefore, very useful at that pitch in detecting cases of beginning auditory nerve degeneration. Mr. Eisenhour has perfected a number of other rods, the highest pitch of which is 15,000 double vibrations. Of course a rod of this pitch vibrates for only a few seconds, but still it is useful in determining whether the individual can or cannot hear such a sound.

Another very decided advance which we would like to report is that Mr. Eisenhour has been able to produce forks of the series which we reported last year and the year before, which are almost overtone-free. Practically every fork when struck emits a number of overtones. These are sometimes very loud and last a long time, and therefore give confusing results, especially as regards the patient's observation of the sound he is expected to hear. As you all know, weights are supplied with the lowest pitched forks and these eliminate the overtones. With the medium pitched forks, however, weights as a rule are not applied except in some of the German forks of the Edelmann series. When these weights are removed, very loud overtones are heard. With the medium pitched forks it is possible to eliminate most of these overtones by applying a piece of rubber tubing around each prong, and while this does not diminish the vibration period of the fork very materially, still it does interfere in a slight degree. The production of these new forks (which have an overtone lasting only about one second or a second and a half) is of great advantage, except that their cost is somewhat increased. It is necessary to have only one or two medium pitched forks, so that this slight increase will not be of material amount.

Your committee believes that these two items, namely, the production of this rather comprehensive series of sounding rods, and the construction of overtone-free medium pitched tuning forks, mark a very important advance in our instrumentarium, and therefore widen our means of making good functional tests of hearing.

The committee recommends the adoption of this report and the continuance of the Committee for the Standardization of Tuning Forks and Hearing Tests.

Respectfully submitted,

L. W. Dean,
E. G. Gill,
Geo. W. Mackenzie,
Robert Sonnenschein,
Chairman.

On motion of Dr. W. L. Benedict, this report was adopted and the committee continued.

Dr. Joseph C. Beck announced that the Committee on Lye Legislation had no report to make.

Report of Senior Member of Council.
1931

The Council submits the following report:

The Council recommends that Dr. Joseph C. Beck be made an Honorary Member of the Academy.

The Council recommends that Dr. J. W. Scales of Pine Bluff, Arkansas; Dr. John Westley Wright of Columbus, Ohio; Dr. W. E. Dixon, of Oklahoma City, Okla.; Dr. Lewis E. Maire, Detroit, Mich.; Dr. F. Vinsonhaler, Little Rock, Ark., and Dr. H. Stillson, Seattle, Wash., be made Life Members of the Academy.

The Council wishes to express to the Research Fellows, Drs. Kirby and McNally, their sincere appreciation for the very excellent work which these Fellows have done during the past year.

The following appropriations for research are recommended: To Dr. Daniel B. Kirby, Research Fellow in Ophthalmology, and to Dr. W. J. McNally, Research Fellow in Otolaryngology, each a stipend of $2,000 for one year.

As supplementary contributions the Council recommends that $500 be given to the Department of Ophthalmology, to be divided as follows: $200 to the International Committee on the Prevention of Trachoma; $300 to the Committee on Physiologic Optics.

The Council is trying to administer the Research Fund as economically as possible. The idea is that the money contributed for research should be only such money as interest on the research fund will permit. With the reorganization of the Academy's affairs this year there has been some change, but this will be taken care of in the future by certain changes in the by-laws which will provide definitely for the amount of money to be expended for research.

The Council recommends that $500 be given to the Department of Otolaryngology, this fund to be distributed under the supervision of Dr. Mosher.

The Council also recommends a contribution of $500 to the Department of Ophthalmic Pathology, as heretofore.

By resolution, the Council has approved the request of Dr. Shurly that the Academy join with other national otolaryngologic societies in the formation of a Library of Otolaryngology. Dr. Joseph C. Beck was named to represent the Academy on this committee.

Dr. W. L. Benedict was named on the Committee on Physiologic Optics.

There has been reorganization, as you know, of the Academy's affairs, and this will necessitate quite a few changes in the constitution and by-laws. The Council, therefore, is giving the usual notice to its Fellows of these proposed changes. These by-laws will appear in the program sent out next year, as usual, thirty days before the meeting of the Academy, so the members can thoroughly digest the proposed changes. Action on these changes will officially take place at the next annual business meeting.

The Council also approves of the Washington bi-centenary activities in the year 1932.

The following officers have been named for the ensuing year:

President	Dr. S. Hanford McKee	Montreal, Canada
President-Elect	Dr. Burt R. Shurly	Detroit, Mich.
1st Vice-President	Dr. Gordon B. New	Rochester, Minn.
2nd Vice-President	Dr. Albert LaSalle	Montreal, Canada
3rd Vice-President	Dr. Gordon F. Harkness	Davenport, Iowa
Comptroller	Dr. Secord H. Large	Cleveland, Ohio
Editor Transactions	Dr. A. W. Proetz	St. Louis
Associate Editor	Dr. Lawrence T. Post	St. Louis
Executive Secretary-Treas.	Dr. W. P. Wherry	Omaha, Neb.
Secretary for Ophthalmology	Dr. W. L. Benedict	Rochester, Minn.
Secretary for Otolaryngology	Dr. J. L. Myers	Kansas City, Mo.
Sec'y Instruction in Ophthalmology	Dr. Harry S. Gradle	Chicago, Ill.
Sec'y Instruction in Otolaryngology	Dr. William V. Mullin	Cleveland, Ohio
Member of Council	Dr. Sylvester J. Beach	Portland, Me.
Member Board of Ophthalmic Exam.	Dr. F. Phinizy Calhoun	Atlanta, Ga.

Place of meeting for 1932: Montreal, Canada.

LUTHER C. PETER.

Moved by Dr. Harry S. Gradle that the report be adopted as a whole, and that the President be instructed to cast the unanimous ballot of the Academy for the officers named. Motion seconded by Dr. T. E. Carmody, and carried.

NEW BUSINESS

DR. LUTHER C. PETER: Dr. Wherry asked us to express our opinion as to our activities this year. I would like to suggest that in subsequent years our arrangements be such that the meetings will not conflict with each other. I was exceedingly anxious to attend Dr. Ames' lecture tonight, but it was impossible to do so and be here. I believe it would be better for the Academy if the different affairs were not so close, a little more time left between our functions, so that our scientific sessions will not be crowded, if a little more care were exercised in separating more widely the various functions which the members wish to attend.

THE PRESIDENT: The members of the various committees on program-making will please take note of that, and if there is no objection we will look upon it as a warning.

DR. HARRY S. GRADLE: I move a vote of thanks of the members of the Academy to the Indiana members who have contributed so much to make this a memorable occasion. (Motion seconded and carried by rising vote.)

DR. W. P. WHERRY: I would move a vote of thanks to Mr. Fawcett, of the French Lick Springs Hotel, for the courteous help he has given us in putting over this meeting.

DR. HARRY S. GRADLE: I would like to second that for the Section on Instruction. Without the active aid of the hotel employees it would have been impossible to have put on the program we did this year. (Motion unanimously carried.)

THE PRESIDENT: Concerning the Indiana physicians, I wish to say that I have never worked with a bunch of boys and girls who were so willing and anxious to please the men who have come to the Hoosier State.

Election of Members

DR. W. P. WHERRY: The following applicants have been elected to membership in the Academy:

Aebli, Rudolf, 30 East 40th St., New York, N. Y., ALR.

Albright, George Carter, 102 S. Clinton St., Iowa City, Iowa, ALR.

Alfaro, Victor Ricardo, 1801 Eye St., N. W., Washington, D. C., ALR.

Arbuckle, David Stuart, Henry Ford Hospital, Detroit, Mich., ALR.

Baer, Benjamin F., Jr., 1927 Spruce St., Philadelphia, Pa., Op.
Barton, John F., Long View, Washington, ALR.
Beisbarth, Carl Conrad, 3720 Washington Ave., St. Louis, Mo., Op.
Berliner, Milton Lionel, 37 W. 57th St., New York, N. Y., Op.
Bernstein, Philip Frank, 1571 Eastern Parkway, Brooklyn, N. Y., ALR.
Beyer, Theodore Ernest, 419 Majestic Bldg., Denver, Colo., ALR.
Black, W. Byron, 535 Professional Bldg., Kansas City, Mo., ALR.
Blackmar, Francis Burton, Woolworth Bldg., Columbus, Ga., ALR.
Bowers, Chester Herbert, 1136 West 6th St., Los Angeles, Cal., ALR.
Boyce, William Alexander, 1210 Roosevelt Bldg., Los Angeles, Cal., Op.
Bricker, Sacks, 3701 N. Broad St., Philadelphia, Pa., ALR.
Bryant, Ben L., 19 Garfield Place, Cincinnati, Ohio, ALR.
Buck, Clair Aurelius, 207 Rogers Bldg., Glens Falls, N. Y., ALR.
Buckingham, Tracy Willis, Bismarck, N. D., ALR.
Bullwinkel, H. Griffin, 30 East 76th St., New York, N. Y., ALR.
Buonaguro, Michael James, 589 Lorimer St., Brooklyn, N. Y., Op.
Buzzard, Josiah Floyd, 1110 Thirteenth Ave., Altoona, Pa., Op.
Cassidy, Waldron Alvin, 1620 Medical Arts Bldg., Omaha, Neb., ALR.
Castroviejo, Ramon, 231 West Washington St., Chicago, Ill., Op.
Chaffers, William Henry, 190 Bates St., Lewiston, Me., ALR.
Clark, Cecil Pratt, 921 Hume-Mansur Bldg., Indianapolis, Ind., Op.
Chamberlain, Webb Parks, 7405 Detroit Ave., Cleveland Ohio.
Croissant, Charles Augustus, 390 Main St., Worcester, Mass., ALR.
Crume, John James, 320 Amarillo Bldg., Amarillo, Texas, ALR.
Davis, Thomas Carroll, 3128 N. Broad St., Philadelphia, Pa., ALR.
Dearmin, Robert Mason, 915 Hume-Mansur Bldg., Indianapolis, Ind., ALR.
De Coste, Stephen Herbert, 1179 Dean St., Brooklyn, N. Y., ALR.
Delaney, James Henry, 138 West 9th St., Erie, Pa., Op.
Denison, Ward C., 20 E. 53rd St., New York, N. Y., ALR.
Dickson, James Charles, 622 Medical Arts Bldg., Houston, Texas, ALR.

Donohoe, Edward Cyril, 306 Professional Bldg., Glendale, Cal., ALR.

Dougherty, Clifford L., 104 S. Michigan Ave., Chicago, Ill., ALR.

Dow, Julian Neal, 727 W. 7th St., Los Angeles, Cal., Op.

Duke, Roncie Renfro, 708 Citizens' Bank Bldg., Tampa, Fla., ALR.

Dunphy, Edwin Blakeslee, 520 Commonwealth Ave., Boston, Mass., ALR.

Earnest, Clarence E., Corwin Hospital, Pueblo, Colo., ALR.

Edgerton, Ambrose Earl, 525 N. Rexford Drive, Beverly Hills, Cal., Op.

Ellis, Bert Edward, 303 Hume-Mansur Bldg., Indianapolis, Ind., ALR.

Equen, Murdock Sykes, 1207 Medical Arts Bldg., Atlanta, Ga., ALR.

Feaster, Henry Joseph, 461 77th St., Brooklyn, N. Y., ALR.

Fewell, Alexander Garrison, 1924 Pine St., Philadelphia, Pa., Op.

Fischbach, Victor William, 2600 Union Central Bldg., Cincinnati, O., ALR.

Flock, Herman F. W., 416 Pine St., Williamsport, Pa., ALR.

Fry, Wilfred Eyles, 1912 Spruce St., Philadelphia, Pa., Op.

Gibby, Harold James, 27 Elm St., Worcester, Mass., ALR.

Giordano, Anthony Joseph, 749 E. 183rd St., New York, N. Y., ALR.

Glatt, Morris A., 3254 Lawrence Ave., Chicago, Ill., ALR.

Goad, Robley Roosevelt, 404 Laurel Bldg., Muscatine, Iowa, Op.

Goldenburg, Michael, 104 S. Michigan Ave., Chicago, Ill., Op.

Goodman, Thomas Lawrence, 1413 Medical Arts Bldg., Fort Worth, Texas, ALR.

Gough, Roy H., 205 Medical Arts Bldg., Memphis, Tenn., ALR.

Harris, J. Harley, Medical Arts Bldg., Memphis, Tenn., ALR.

Haupt, William Carl, 1235 Dean St., Brooklyn, N. Y., ALR.

Heinberg, Charles Jerome, 404 Blount Bldg., Pensacola, Fla., ALR.

Hendricks, Louis Joseph, 210 W. 12th St., Cincinnati, Ohio, Op.

Hewson, William, 1721 Pine St., Philadelphia, Pa., ALR.

High, Isaac Beidler, 326 North 5th St., Reading, Pa., ALR.

Hilding, Anderson C., Mayo Clinic, Rochester, Minn., ALR.

Hollender, Abraham Risel, 30 N. Michigan Ave., Chicago, Ill., ALR.

Holzer, William F., 36 Pleasant St., Worcester, Mass., Op.

Hotchkiss, Walter Thomson, Aladdin Bldg., Miami Beach, Fla., ALR.

Howard, Robert Edwin, 2600 Union Central Bldg., Cincinnati, O., ALR.

Howell, Donald McLean, 1608 Eaton Tower, Detroit, Mich., ALR.

Hubbard, William Barrington, 301 Dryden Bldg., Flint, Mich., ALR.

Hume, John Raymond, 1230 Maison Blanche Bldg., New Orleans, La., ALR.

James, William Mahlon, 640 S. Kingshighway, St. Louis, Mo., Op.

Jameson, Patrick Chalmers, 139 Montague St., Brooklyn, N. Y., Op.

Jennings, Charles W., 315 Highland Bldg., Pittsburgh, Pa., Op.

Jerome, William Ziporkes, 436 Fort Washington Ave., New York, N. Y., ALR.

Kittredge, Frank Everett, Masonic Temple, Nashua, N. H., ALR.

Knighton, Willis Sackett, 40 E. 61st St., New York, N. Y., Op.

Kronfeld, Peter Clemens, 950 E. 59th St., Chicago, Ill., Op.

Kulvin, Max M., 1 N. Crawford Ave., Chicago, Ill., ALR.

Lampert, Max Thaler, 55 E. Washington St., Chicago, Ill., ALR.

Lawson, Lawrence James, 636 Church St., Evanston, Ill., ALR.

Leasure, John Kent, 611 Hume-Mansur Bldg., Indianapolis, Ind., ALR.

Leshin, Norman, 180 N. Michigan Ave., Chicago, Ill., ALR.

Levine, Joseph, 113 East 61st St., New York, N. Y., Op.

Levy, Harry H., 1016 Fifth Ave., New York, N. Y., Op.

Littell, Joseph Jerome, 603 Hume-Mansur Bldg., Indianapolis, Ind., ALR.

Logue, Lyle James, 1304 Walker Ave., Houston, Texas, ALR.

Lowell, William Holbrook, 82 Commonwealth Ave., Boston, Mass., Op.

Luhr, Alfred Francis, 40 West North St., Buffalo, N. Y., Op.

Luong, Romeo A., 1713 Pine St., Philadelphia, Pa., ALR.

Lynch, Robert James, 818 Equitable Bldg., Des Moines, Iowa, ALR.

McAndrews, Leo Francis, 1930 Chestnut St., Philadelphia, Pa., Op.

McDougall, William Lowndes, 417 Atlanta National Bank Bldg., Atlanta, Ga., ALR.

McGregor, Gregor William, 170 St. George St., Toronto, Canada, ALR.

McKeown, Hugh Spencer, 30 West 59th St., New York, N. Y., Op.

MacCready, Paul Beattie, 442 Temple St., New Haven, Conn., ALR.

Meek, Raymond Emory, 30 E. 40th St., New York, N. Y., Op.

Melton, Bernard Lee, Jr., 605 Goodrich Bldg., Phoenix, Ariz.

Moyle, Eugene Henry, 1088 Park Ave., New York, N. Y., ALR.

Miller, Theodore Earl, 122 S. Michigan Ave., Chicago, Ill., ALR.

Miller, William Humphreys, 502 Tribune Bldg., Terre Haute, Ind.

Moss, Olin Winn, 611 Weber Bldg., Lake Charles, La., ALR.

Mueller, Lillian Barbara, 404 Continental Bank Bldg., Indianapolis, Ind., ALR.

Murray, Henry Joseph, 53 South St., Stamford, Conn., ALR.

Newton, William Burton, 201 N. Second Ave., Alpena, Mich, OALR.

Norton, Arthur Holmes, 1162 Willamette St., Eugene, Ore., Op.

O'Hora, Bernard Anthony, 8-266 General Motors Bldg., Detroit, Mich., ALR.

Parker, Harry Lawrence, 88 East Ave., Lockport, N. Y., ALR.

Peer, Lyndon Arthur, 965 Broad St., Newark, N. J., ALR.

Perheram, Chas. Creglaw, 642 Medical Arts Bldg., Portland, Ore., ALR.

Poirier, George Henri, 60 Bay State Road, Boston, Mass., ALR.

Rhein, Alfred Edwin, 414 Tribune Bldg., Terre Haute, Ind.

Rosenbluth, Michael, 1018 E. 163rd St., New York, N. Y., ALR.

Russell, Roland Doyle, 58 E. Washington St., Chicago, Ill., ALR.

Schiefelbein, Harry Theodore, Stevens Clinic Hospital, Welch, W. Va., Op.

Schneck, Nathan, 815 S. 8th St., Manitowoc, Wis., ALR.

Sciarretta, Sylvio Amedeo, 55 East Washington St., Chicago, Ill., ALR.

Seltz, Samuel, Provident Bank Bldg., Cincinnati, Ohio, ALR.

Settel, Nathan, 250 W. 94th St., New York, N. Y., ALR.

Seward, Jackson Anderson, 121 E. 60th St., New York, N. Y., ALR.

Shannon, Clarence William, 1215 5th Ave., Seattle, Wash., Op.

Simpson, George Victor, 1710 R. I. Ave., Washington, D. C., Op.

Smith, James Morrisset, 123 E. 53rd St., New York, N. Y., ALR.

Smyth, Duncan Campbell, 375 Commonwealth Ave., Boston, Mass., ALR.

Snelling, Pinckney Welch, 179 Allyn St., Hartford, Conn., ALR.

Stevenson, William Warren, 416 Kresge Bldg., Flint, Mich., ALR.
Streit, August J., 811 Fisk Bldg., Amarillo, Texas, ALR.
Taylor, Edgar Merle, 642 Medical Arts Bldg., Portland, Ore., Op.
Thorpe, Harvey Edward, 8075 Jenkins Arcade, Pittsburgh, Pa., Op.
Tusak, Ervin Anthony, 115 E. 90th St., New York, N. Y., Op.
Vail, Derrick Tilton, Jr., 24 E. 8th St., Cincinnati, O., Op.
Valone, Frank Horace, 258 Genesee St., Utica, N. Y., ALR.
Van Osdel, Harry Allen, 828 Chamber of Commerce, Indianapolis, Ind., ALR.
Wachsberger, Alfred, 51 W. 73rd St., New York, N. Y., ALR.
White, Vivian Parley, 1010 Ezra Thompson Bldg., Salt Lake City, Utah, ALR.
Wickliffe, Thompson Flournoy, Tweedy Bldg., Jasper, Ala., ALR.
Wilson, Robert G., Kresge Bldg., Batavia, N. Y., ALR.
Wincor, Henry G., 2021 Grand Concourse, New York, N. Y., Op.
Wolcott, Charles Clements, 291 N. Broadway, Yonkers, N. Y., ALR.
Yates, Earl Carroll, 190 N. Upper St., Lexington, Ky., ALR.

DR. W. P. WHERRY: These men have all received their certificates from the Boards and are found to be acceptable to the Council. I move you that this list of 138 names be elected to membership in the Academy. (Motion seconded by Dr. Gradle and carried.)

THE PRESIDENT: I will appoint Dr. Carmody and Dr. Benedict to escort our newly elected President to the platform.

DR. S. HANFORD MCKEE: Mr. President and Members of the Academy—I will detain you only a minute or two, and I would like to reiterate what I said last night about the wonderful feeling it has given me to be elected by this Academy to this high office. I assure you that no one who has ever assumed the office has done so with greater pride, and with a more acute idea of his responsibilities and obligations to this Academy. You know as well as I how the work of the Academy goes on. The Executive Secretary-Treasurer has the work so well in hand that I really feel something like the policeman who was on a traffic corner when a woman driver came along—one of the kind that holds out her hand just the same, whether she wants to stop, to go ahead or to turn right or left. The policeman held up his hand, but she

went right ahead; he shouted at her to stop, and she finally did go in to the curb. He went over to her and said, "Didn't you see me hold up my hand?" "Yes, I saw you." "Didn't you hear me tell you to stop?" "Yes, I heard you." "Well," said the cop, "I might as well go home. I don't seem to be doing much good here." I think that is pretty much the position of the President of this wonderful organization. But I promise you I am not going home—I will hang around.

As regards the meeting next year in Montreal, if it can be held shortly after the middle of September, before our Fall weather sets in, the chances are we will have good weather. I hope you will come. We can promise you a good meeting and a good time —plenty of golf and tennis and anything else you want.

I would like again to say how much I appreciate this honor, not for myself, but for our Canadian members. It is a great tribute to men like Dr. Birkett, who has been the great representative of our specialties in the city of Montreal, as you know, and it is a tribute to all the Canadian members. I thank you more than I can say for this great honor.

THE PRESIDENT: I will ask Dr. Newhart and Dr. Lancaster to bring forward our President-elect.

DR. BURT R. SHURLY: Mr. President and Members—It is too hot tonight to make a speech or tell a story, but an honor of this sort comes only once in a lifetime to any one of us. I look upon this as the greatest of our special societies, and it is indeed a marvelous honor and privilege and pleasure to be able to serve, because as we go through life I think one of the great joys is to have opportunity to serve our profession.

I am sure we will have a marvelous session at Montreal. I have been there several times, and if there is anyone here who has not been there I can give it every recommendation in the world as a marvelous place for a meeting, and the Canadian brand of hospitality is exceeded by none.

What the future will map out for us remains to be seen, but with your loyal support and help, I am sure this Academy will go on to better and finer things. I assure you that from the bottom of my heart I appreciate this honor.

THE PRESIDENT: Gentlemen, we stand adjourned.

SECTION ON OTOLARYNGOLOGY

Friday Afternoon Session

The Friday afternoon session convened at one-thirty, the President, Dr. John F. Barnhill, presiding.

Dr. Ralph A. Fenton (and Dr. Olaf Larsell), Portland, Ore., presented a paper entitled "Some Experimental and Clinical Observations on the 'Reticulo-Endothelial' Components of Accessory Sinus Mucosa." Discussed by Drs. Frank J. Novak, Chicago; Harris P. Mosher, Boston, and Ralph A. Fenton.

Dr. John A. Pratt, Minneapolis, Minn., read a paper entitled "Atrophic Rhinitis." Discussed by Drs. Hal Foster, Kansas City, Mo.; Harry L. Pollock, Chicago, and John A. Pratt.

Dr. Maxwell Fineberg, St. Louis, Mo., read a paper entitled "General Otological Aspects of Cholesterin Metabolism." Discussed by Drs. S. J. Kopetzky, New York City, and Maxwell Fineberg.

Dr. Gabriel Tucker, Philadelphia, read a paper entitled "Early Intrinsic Cancer of the Larynx: Diagnosis and Treatment with Observations in Laryngofissure as a Method of Cure in a Series of Over Twenty-five Cases." Discussed by Drs. Gordon B. New, Rochester, Minn.; Robert Sonnenschein, Chicago; Harry L. Pollock, Chicago; T. E. Carmody, Denver; Joseph C. Beck, Chicago, and Gabriel Tucker

The Thirty-sixth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology adjourned *sine die*.

ALPHABETICAL LIST OF MEMBERS

HONORARY FELLOWS

*Beck, Joseph C. (1901).....185 N. Wabash Ave., Chicago, Ill., ALR
*Birkett, H. S.....1190 Mountain St., Montreal, Canada, ALR
Dundas-Grant, Sir James.....148 Harley St., W. 1, London, Eng.
Elliot, Lt.-Col. R. H.
54 Welbeck St., Cavendish Sq., W., London, Eng.
Elschnig, Prof. A., Ferdinandstrasse, 10, Prague, Czecho-Slovakia
Graves, Basil
11 Devonshire Place, Upper Wimpole St., W. 1, London, Eng.
Gray, Albert A.....Glasgow, Scotland
Grósz, Prof. Dr. Emil de.....Budapest, VIII, Maria, V. 39, ALR
*Jackson, Edward (1902).....314 14th St., Denver, Colo., OP
Johnson, Lindsay, Britannia Bldg., West St., Durban, Natal, Africa
Kirkpatrick, Lt.-Col. Henry, 54 Welbeck St., W. 1, London, Eng.
Lawford, J. B.....Sagamore, New Road, Ashtead, Surrey, Eng.
Lister, Sir William T.....24 Devonshire Place, W. 1, London, Eng.
Nagar, Prof F.....Freiestrasse 20, Zurich, Switzerland
Tilley, Herbert.....University College Hospital, London, Eng., Op.
Van der Hoeve, Prof. J.....Rijnsburgerweg, 6 A, Leiden, Holland

LIFE MEMBERS

Allen, J. H.....Republic Bldg., Denver, Colo., ALR
Brown, Edward J. (1911)
900 Nicollet Ave., Minneapolis, Minn., OALR
Day, Ewing W.....121 University Place, Pittsburgh, Pa., OALR
Dixon, W. E.....Medical Arts Bldg., Oklahoma City, Okla., OALR
*Foster, Hal (1894)
1426 Professional Bldg., Kansas City, Mo., ALR
Kollock, C. W. (1905)..86 Wentworth St., Charleston, S. C., OALR
Lochard, L. B.....Metropolitan Bldg., Denver, Colo., OALR
Maire, Lewis E. (1906).....Kresge Bldg., Detroit, Mich., OALR
Mayer, Emil (1905), 320 W. 89th St., New York City, N. Y., ALR
Owen, Frank S.....Medical Arts Bldg., Omaha, Nebr. OALR
Posey, Wm C.....Radnor, Pa., Op.
Scales, J. W. (1896).....204½ Main St., Pine Bluff, Ark., OALR
Schneideman, T. B. (1904)
2045 Chestnut St., Philadelphia, Pa., Op.
Selfridge, G. L.....384 Post St., San Francisco, Cal., ALR
Stein, O. J.....Palos Verdes Estates, Calif., ALR

STEWART, T. M.....901 Union Trust Bldg., Cincinnati, O., OALR
STILLSON, H.....Seaboard Bldg., Seattle, Wash., OALR
VINSONHALER, F. (1902)....Urquhart Bldg., Little Rock, Ark., OALR
WOOD, CASEY A.....7 W. Madison St., Chicago, Ill., Op.
WRIGHT, JOHN W.....137 E. State St., Columbus, Ohio, Op.
*YOUNG, H. B.....204 Jefferson St., Burlington, Ia., Op.A

*Attended 1931 meeting. Number in parentheses indicate year in which the Fellow became a member of the Academy. Fellows are requested to supply missing data and report errors in addresses or specialties to the Executive Secretary, Dr. W. P. Wherry, Medical Arts Bldg., Omaha, Nebr.

FELLOWS

A

ABBOTT, W. J. (1919)..Carnegie Medical Bldg., Cleveland, O., OALR
*ABRAHAMSON, IRA A. (1927)
605 Traction Bldg., Cincinnati, O., Op.
ADAMS, A. L. (1905).....Professional Bldg., Jacksonville, Ill., OALR
ADAMS, C. F. (1908).....34 West State St., Trenton, N. J., OALR
*ADAMS, CHARLES JOSEPH (1928)
101 W. Sycamore St., Kokomo, Ind., OALR
*ADAMS, D. S.....508 Hume-Mansur Bldg., Indianapolis, Ind., OALR
ADAMS, ELDRIDGE S. (1920)
Aeronautics Branch, Dept. of Commerce, Washington, D. C., ALR
ADAMS, FRANK M. (1924)
122 Waterman St., Providence, R. I., ALR
ADAMS, FRED'K W. (1920)
Roosevelt Clinic, 1114 Boylston Ave., Seattle, Wash., OALR
ADKINS, GEO. ELI (1920)..Jackson Infirmary, Jackson, Miss., ALR
ADLER, FRANCIS HEED (1920)
313 S. 17th St., Philadelphia, Pa., Op.
AEBLI, RUDOLF (1931).....30 East 40th St., New York, N. Y., Op.
AGAN, WM B. (1930).....510 Third St., Brooklyn, N. Y., Op.
AGNEW, F. F. (1920)....120 3rd Ave., S. E., Independence, Ia., OALR
AINSLIE, GEORGE (1921).....Selling Bldg., Portland, Ore., OALR
ALBRIGHT, LT.-COM. P. M., M. C. U. S. Navy (1922)
U. S. Naval Hospital, Annapolis, Md., OALR
ALBRIGHT, GEO C. (1931)..102 S. Clinton St., Iowa City, Ia., ALR
*ALDEN, ARTHUR M. (1922).....Frisco Bldg., St. Louis, Mo., ALR
ALDERDYCE, WM. (1908).....513 Madison Ave., Toledo, O., OALR
ALEXANDER, GEO. J.....1930 Chestnut St., Philadelphia, Pa., ALR
ALFARO, V. R. (1931)
1801 Eye St., N. W., Washington, D. C., ALR
ALKIRE, H. L. (1898).....614 Kansas Ave., Topeka, Kan., OALR
ALLEN, CHAS E. (1920)
712 Waldheim Bldg., Kansas City, Mo., OALR
ALLEN, G. HENRY (1916).....Mills Bldg., Topeka, Kan., OALR

ALLEN, G. V. (1921).....835 Kansas Ave., Topeka, Kan., OALR
*ALLEN, ORRIS T. (1928)
422 Rose Dispensary Bldg., Terre Haute, Ind., OALR
*ALLEN, THOMAS DYER (1928)
122 S. Michigan Ave., Chicago, Ill., Op.
ALMOUR, RALPH W. (1925)
51 W. 73rd St., New York, N. Y., ALR
ALTER, F. W.....219 15th St., Toledo, O., OALR
ALTRINGER, A. N. (1920)
Professional Bldg., Kansas City, Mo., ALR
ALVIS, BENNETT Y. (1928)
900 Carleton Bldg., St. Louis, Mo., Op.
AMANO, K. W. (1929), 502 Woodland Ter., Philadelphia, Pa., ALR
AMBERG, EMIL.....662 Maccabee Bldg., Detroit, Mich., ALR
AMBROSE, ELMER C. (1927).....910½ Main St., Trenton, Mo., OALR
ANDERSON, CARL M. (1926).....Mayo Clinic, Rochester, Minn., ALR
ANDERSON, W. B.....Med. Arts Hosp., Brownwood, Tex., OALR
*ANDREWS, A. H. (1896).....30 N. Michigan Ave., Chicago, Ill., OALR
ANDREWS, B. F. (1912).....25 E. Washington St., Chicago, Ill., OALR
ANDREWS, H. D. (1912).....33 Allen St., Buffalo, N. Y., Op.
ANDREWS, M. P. (1915).....926 S. 8th St., Manitowoc, Wis., OALR
ANNON, W. T.....235 S. 15th St., Philadelphia, Pa., ALR
ANTHONY, DANIEL HARBERT (1929)
Exchange Bldg., Memphis, Tenn., Op.
APPLEMAN, L. F. (1913)
308 South 16th St., Philadelphia, Pa., Op.
ARBUCKLE, DAVID S. (1931)
Henry Ford Hospital, Detroit, Mich., ALR
*ARBUCKLE, MILLARD F.
1011 Beaumont Med. Bldg., St. Louis, Mo., ALR
*ARMSTRONG, R. M. (1928)
Fayette Bank Bldg., Lexington, Ky., OALR
ARNOLD, FRANCIS J.....182 Pearl St., Burlington, Vt., OALR
*ARONSON, SIDNEY S. (1929)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
ARRELL, W.....317 Main St., E. Hamilton, Ontario, OALR
*ASBURY, MARY K. (1925).....3549 Holly Lane, Cincinnati, O., Op.
ASH, GARRETT G. (1920).....1 Main St., Bradford, Pa., OALR
ASHLEY, T. W. (1920).....5711 8th Ave., Kenosha, Wis., OALR
ATKINSON, D. T. (1918)
827 Med. Arts Bldg., San Antonio, Tex., OALR
ATKINSON, THOMAS E. (1929)
Coronado Bldg., Greeley, Colo., ALR
*ATKINSON, WALTER S. (1925)
168 Sterling St., Watertown, N. Y., Op.
AUERBACH, JULIUS.....1016 Fifth Ave., New York City, N. Y., ALR
AYNESWORTH, H. T. (1912), Medical Arts Bldg., Waco, Tex., OALR

B

*Babbitt, James A. (1924), 1912 Spruce St., Philadelphia, Pa., ALR
Baer, Benjamin F., Jr. (1931)
1927 Spruce St., Philadelphia, Pa., Op.
Baeseman, R. Winfield (1929)
Jersey Central Bldg., Asbury Park, N. J., ALR
*Bahn, Chas A. (1922)
1707 Pere Marquette Bldg., New Orleans, La., Op.
Bailey, Fred W. (1912)
Merchants Nat'l Bk. Bldg., Cedar Rapids, Ia., OALR
Bailey, Harold (1908), 840 Landers Bldg., Springfield, Mo., OALR
Baird, Chas. G. (1920), 301 E. Chapel St., Santa Maria, Cal., OALR
Baker, Chas H.....Bay City Bk. Bldg., Bay City, Mich., OALR
Baker, Harry B. (1916).....59 Main St., Taunton, Mass., OALR
*Baldwin, H. E. (1930).....Temple Bldg., Danville, Ill., ALR
Baldwin, Kate W.....1117 Spruce St., Philadelphia, Pa., ALR
Balentine, P. L. (1915), 1524 Chestnut St., Philadelphia, Pa., Op.
Ball, Michael V.....316 Hazel St., Warren, Pa., OALR
*Ballenger, H. C. (1915), 25 E. Washington St., Chicago, Ill., ALR
Ballin, M. J.....20 E. 76th St., New York City, N. Y., ALR
Ballon, D. H. (1924).....1471 Crescent St., Montreal, Can., ALR
Bane, Wm. C.....1005 Republic Bldg., Denver, Colo., OALR
Bane, W. M.....1005 Republic Bldg., Denver, Colo., OALR
Barber, F. (1913).....75 S. Fitzhugh St., Rochester, N. Y., Op.
Bardenheier, F. G. A. (1909).....Frisco Bldg., St. Louis, Mo., ALR
Barkan, Adolph.....490 Post St., San Francisco, Cal., OALR
Barkan, Hans.....490 Post St., San Francisco, Cal., Op.
Barkan, Otto (1929).....490 Post St., San Francisco, Cal., Op.
Barker, Chas. B. (1920)
224½ W. Okla. Ave., Guthrie, Okla., OALR
Barker, O. G. A. (1909)
804 Johnstown Trust Co., Johnstown, Pa., Op.
Barlow, Roy A.....Hiram Sibley Bldg., Rochester, N. Y., ALR
Barnett, Saul E. (1929).....25 Parson St., Detroit, Mich., ALR
*Barnhill, J. F. (1896)
5369 Pine Tree Drive, Miami Beach, Fla., ALR
Barton, John F. (1931) .....Longview, Wash., ALR
*Bass, A. L. (1924).....Heyburn Bldg., Louisville, Ky., OALR
Bassen, Ed. J. (1930).....698 Madison Ave., New York, N. Y., Op.
Baum, H. L. (1917).....510 Republic Bldg., Denver, Colo., ALR
Baum, Wm. W. (1930).....Guardian Bldg., Salem, Ore., ALR
Beach, B. S. (1927).....140 W. 86th St., New York, N. Y., Op.
*Beach, S. J. (1914).....704 Congress St., Portland, Me., Op.
Beal, Homer A. (1926), 1002 Argyle Bldg., Kansas City, Mo., ALR
*Beals, C. Wearne (1930).....41 No. Brady St., DuBois, Pa., ALR
*Beard, Harold Jolly (1924),
510 Dollar Bank Bldg., Youngstown, O., OALR
*Beattie, Robert (1906),
David Whitney Bldg., Detroit, Mich., OALR

BEATTIE, WILLIAM H. (1921), 258 Genesee St., Utica, N. Y., OALR
*BEATTY, H. G.....681 E. Broad St., Columbus, O., ALR
BEAUDOUX, HARRY A.....230 Grand Ave., Oakland, Cal., OALR
*BECK, A. L. (1927)....421 Huguenot St., New Rochelle, N. Y., OALR
*BECK, FREDERICK L. (1920),
408 Hynds Bldg., Cheyenne, Wyo., ALR
BECKER, H. M.....49 S. 4th St., Sunbury, Pa., OALR
*BEDELL, ARTHUR J.....344 State St., Albany, N. Y., Op.
BEEBE, C. S.....120 E. Wisconsin Ave., Milwaukee, Wis., Op.
*BEEM, IONE F. (1916).....31 N. State St., Chicago, Ill., OALR
BEGLE, HOWELL LLEWELLYN (1928),
2730 E. Jefferson Ave., Detroit, Mich., Op.
BEIDEMAN, J. E. (1929),
Montgomery Trust Arcade, Norristown, Pa., Op.
BEIL, J. W. (1918).....Bryant Bldg., Kansas City, Mo., OALR
*BEISBARTH, CARL C. (1931),
3720 Washington Ave., St. Louis, Mo., Op.
BELL, M. H. (1913), First Nat'l Bank Bldg., Vicksburg, Miss., Op.
BELL, WILL OTTO (1928),
1431 Medical Dental Bldg., Seattle, Wash., Op.
BELLIN, JOSEPH.....523 W. 6th St., Los Angeles, Cal., OALR
BELLOWS, GEORGE E. (1896),
Professional Bldg., Kansas City, Mo., Op.
BENDER, H. A. (1930).....Black Bldg., Waterloo, Ia., ALR
BENDER, M. S. (1930).....11 150th St., Jamaica, N. Y., ALR
Also 30 E. 40th St., New York, N. Y.
*BENEDICT, WILLIAM L. (1919), Mayo Clinic, Rochester, Minn., Op.
*BENNETT, ARTHUR L.....150 N. Pearl St., Buffalo, N. Y., Op.
BENTLEY, NEIL L., 1161 David Whitney Bldg., Detroit, Mich., OALR
*BERENS, CONRAD (1924), 35 E. 70th St., New York City, N. Y., Op.
BERLINER, MILTON L. (1931),
57 W. 57th St., New York, N. Y., Op.
BERNATZ, C. F.....Park Bldg., Pittsburgh, Pa., Op.
BERNHEIMER, L. BENNO (1930),
104 S. Michigan Ave., Chicago, Ill., ALR
BERNSTEIN, E. J. (1900).....10 Peterboro, Detroit, Mich., OALR
BERNSTIEN, PHILIP F. (1931)
1571 Eastern Parkway, Brooklyn, N. Y., ALR
BERRISFORD, PAUL D.....Lowry Medical Bldg., St. Paul, Minn., OALR
*BERRY, D. F. (1911),
710 Hume-Mansur Bldg., Indianapolis, Ind., ALR
BERRY, GORDON.....36 Pleasant St., Worcester, Mass., ALR
*BEYER, A. G. (1925).....Doctors' Bldg., Cincinnati, O., OALR
BEYER, LOUIS J. (1918)....449 Delaware Ave., Buffalo, N. Y., ALR
BEYER, THEODORE E. (1931).....Majestic Bldg., Denver, Colo., ALR
BINGER, H. E. (1917)..Lowry Medical Bldg., St. Paul, Minn., OALR
BINKLEY, ROY S. (1915).....Fidelity Bldg., Dayton, O., OALR
*BIRSNER, LOUIS J. (1926).....Missouri Bldg., St. Louis, Mo., ALR
BLAAUW, E. E.....190 Ashland Ave., Buffalo, N. Y., Op.

BLACK, MELVILLE (1904),
424 Metropolitan Bldg., Denver, Colo., Op.
*BLACK, NELSON MILES.....Huntington Bldg., Miami, Fla., Op.
BLACK, PAUL (1927).....Stuart Bldg., Lincoln, Neb., Op.
BLACK, W. BYRON (1931),
Professional Bldg., Kansas City, Mo., ALR
BLACK, W. D. (1900).....Metropolitan Bldg., St. Louis, Mo., ALR
BLACKBURN, W. J. (1919).....663 Reibold Bldg., Dayton, O., OALR
*BLACKMAR, FRANCIS B. (1931),
Swift Kyle Bldg., Columbus, Ga., ALR
BLAIR, MORTIMER WARREN (1928),
N. E. cor. 20th and Chestnut Sts., Philadelphia, Pa., Op.
BLAIR, W. W.....121 University Place, Pittsburgh, Pa., Op.
*BLAKESLEY, T. S.....Professional Bldg., Kansas City, Mo., OALR
BLASSINGAME, C. D. (1924),
Physicians and Surgeons Bldg., Memphis, Tenn., ALR
*BLEDSOE, R. W. (1908), 1005 Madison Ave., Covington, Ky., OALR
*BLICKENSTAFF, A. J. (1922).....Peoria Life Bldg., Peoria, Ill., OALR
BLUE, J. B. (1916),
Columbian Mutual Tower Bldg. Memphis, Tenn., OALR
BLUM, HENRY N.
1211 Maison Blanche Bldg., New Orleans, La., Op.
BOBB, E. V. (1918),
101 First Nat'l Bank Bldg., Mitchell, S. D., OALR
BOECKMANN, EGIL.....Medical Arts Bldg., St. Paul, Minn., OALR
BOEHRINGER, W. H.,
700 Central Medical Bldg., Philadelphia, Pa., OALR
*BOEMER, L. C. (1930), 3720 Washington Blvd., St. Louis, Mo., ALR
BOERNER, MORRIS H. (1923).....Norwood Bldg., Austin, Tex., OALR
BOILER, W. F. (1912).....Dey Bldg., Iowa City, Ia., OALR
BOLOTOW, NATHAN A. (1929),
108 Waterman St., Providence, R. I., ALR
BONE, HERMAN D.....19 Pleasant St., Gardner, Mass., OALR
BONNER, WM. F. (1926),
Medical Arts Bldg., Wilmington, Del., OALR
BOONE, J. C (1921).....Sherland Bldg., South Bend, Ind., ALR
BOONE, J. L. (1924).....Professional Bldg., Jacksonville, Fla., ALR
BORDLEY, JAMES, JR.....330 N. Charles St., Baltimore, Md., OALR
BORGESON, E. J. (1930),
Medical Arts Bldg., Minneapolis, Minn., Op.
*BOTHMAN, LOUIS (1930), 122 S. Michigan Ave., Chicago, Ill., Op.
BOUCHER, R. B.....414 Birks Bldg., Vancouver, B. C., OALR
BOURBON, OLIVER P. (1920),
520 W. Seventh St., Los Angeles, Cal., OALR
BOUSQUET, P. E. (1924),
66 Sherbrooke St., W., Montreal, Can., OALR
BOUVY, HARRY M. (1920)..385½ Washington, Portland, Ore., OALR
BOUVY, L. B. (1927).....Foley Bldg., La Grande, Ore., OALR
*BOWERS, CHESTER H. (1931),
1136 West 6th St., Los Angeles, Cal., ALR

BOYCE, G. H. (1912),
First Nat'l Bank Bldg., Iron Mountain, Mich., OALR
BOYCE, W. B. (1912)....1107 Ludington St., Escanaba, Mich., OALR
*BOYCE, WM. A. (1931).....Roosevelt Bldg., Los Angeles, Cal., Op.
BOYD-SNEE, HARRY.....716 J. M. S. Bldg., South Bend, Ind., ALR
BOZER, HERRMANN EUGENE (1923),
468 Delaware Ave., Buffalo, N. Y., ALR
BRASWELL, JAMES C. (1922)
1109 Medical Arts Bldg., Tulsa, Okla., OALR
BRAUNLIN, WM. H. (1920),
Marion Nat'l Bank Bldg., Marion, Ind., OALR
BRAWLEY, F. E.....30 N. Michigan Blvd., Chicago, Ill., Op.
BRAWNER, LEON ED. (1929),
157 Forrest Ave., N. E., Atlanta, Ga., ALR
BRAY, E. R. (1912), 342 Lowry Medical Bldg., St. Paul, Minn., OALR
BREDLAU, ERNEST A. (1929)....2753 North Ave., Chicago, Ill., ALR
BREHMER, HARRISON L. (1920),
First Nat'l Bank Bldg., Albuquerque, N. M., OALR
BREWER, JOHN D. (1920),
Baird Brewer General Hospital, Dyersburg, Tenn., OALR
*BRIANT, THOMAS E. (1929).....28 E. Main St., Welland, Ont., ALR
BRIBACH, EUGENE J. (1919), Simpson Bldg., Atchison, Kan., OALR
BRICKER, SACKS (1931), 3701 N. Broad St., Philadelphia, Pa., ALR
BRICKLEY, DANIEL W. (1918)
Uhler Phillips Bldg., Marion, O., OALR
BRIGGS, F. W.....Havre, Mont., OALR
BRIGGS, W. E.....1027 10th St., Sacramento, Cal., OALR
BRITTEN, G. S. (1907)....Medical Arts Bldg., Syracuse, N. Y., ALR
BROBST, C. H. (1907)....Central Nat'l Bank Bldg., Peoria, Ill., OALR
BRODRICK, FRANK W.....Central Trust Bldg., Sterling, Ill., OALR
BROOKS, EARL B. (1921).....939 Stuart Bldg., Lincoln, Neb., OALR
BROOKS, E. H. (1915)....108 W. College Ave., Appleton, Wis., OALR
BROSE, L. D.....201 South East First St., Evansville, Ind., OALR
*BROWN, ALBERT LOUIS (1926), Doctors Bldg., Cincinnati, O., Op.
BROWN, AUDREY O. (1927),
742 Maccabee Bldg., Detroit, Mich., OALR
BROWN, C. M.....510 Delaware Ave., Buffalo, N. Y., ALR
BROWN, CHARLES WILLIAM (1927),
Medico-Dental Bldg., San Diego, Cal., ALR
BROWN, CLYDE O. (1928),
3720 Washington Ave., St. Louis, Mo., ALR
BROWN, H. ALEXANDER (1912)
291 Geary St., San Francisco, Cal., Op.
BROWN, JOHN C. (1920), Medical Arts Bldg., St. Paul, Minn., OALR
BROWN, JOHN E. (1901)....370 East Town St., Columbus, O., OALR
BROWN, J. M.....1136 W. 6th St., Los Angeles, Cal., ALR
BROWN, L. E. (1917).....2nd Nat'l Bank Bldg., Akron, O., OALR
BROWN, MORROW D.....432 Republic Bldg., Denver, Colo., ALR
BROWN, MORTIMER G. (1921),
604 Medical Arts Bldg., Syracuse, N. Y., ALR

BROWN, SAMUEL HORTON (1910),
1930 Chestnut St., Philadelphia, Pa., Op.
BROWN, W. HAROLD (1912),
Bank of Montreal Bldg., Edmonton, Alberta, Can., OALR
BROWNELL, MORTON EMMONS (1928),
101 N. Main, Wichita, Kan., Op.
BRUCE, G. M. (1930),
180 Fort Washington Ave., New York, N. Y., Op.
BRUDER, JOSEPH (1908), 57 W. 57th St., New York City, N.Y., OALR
BRUERE, GUSTAVE E. (1919),
Medical Dental Bldg., Portland, Ore., OALR
BRUMM, SETH A. (1922),
Medical Arts Bldg., Philadelphia, Pa., OALR
BRUNER, ABRAM BRENEMAN (1922),
1214 Guardian Bldg., Cleveland, O., OALR
*BRUNER, WM. E.....Guardian Bldg., Cleveland, O., Op.
BRUST, H. O (1913).....820 S. Crouse Ave., Syracuse, N. Y., ALR
BRYAN, JAMES LESLIE (1928),
919 Bennie-Dillon Bldg., Nashville, Tenn., OALR
BRYANT, ALICE G. (1908).....502 Beacon St., Boston, Mass., ALR
BRYANT, BEN L. (1931).....19 Garfield Place, Cincinnati, O., ALR
BUCHANAN, NORMAN D. (1925)
479 Water St., Peterborough, Ont., Canada, OALR
BUCK, CLAIR A. (1931).....Rogers Bldg., Glens Falls, N. Y., ALR
BUCKINGHAM, TRACY W. (1931).....Bismarck, N. Dak., ALR
BUCKLEY, J. H. (1908)..1st Nat'l Bank Bldg., Ft. Smith, Ark., OALR
BUCKMAN, E.U. (1906), 70 S. Franklin St.,Wilkes-Barre, Pa., OALR
BULLWINKEL, H. GRIFFIN (1931)
30 East 76th St., New York, N. Y., ALR
*BULSON, A. E. (1896).....406 W. Berry St., Ft. Wayne, Ind., OALR
*BULSON, EUGENE LORING (1928),
406 W. Berry St., Ft. Wayne, Ind., OALR
BUONAGURO, MICHAEL J. (1931),
589 Lorimer St., Brooklyn, N. Y., Op.
BURCH, FRANK E. (1907),
Miller Hospital Clinic, St. Paul, Minn., Op.
BURDICK, AUSTIN F. (1925),
330 N. Capitol Ave., Lansing, Mich., OALR
BURKE, JOHN R. (1927).....207 Elm St., Holyoke, Mass., OALR
BURKE, T. A.....Rose Bldg., Cleveland, O., OALR
BURLESON, J. H. (1917),
1034 Nix Professional Bldg., San Antonio, Tex., OALR
BURNS, L. J. (1930).....1930 Chestnut St., Philadelphia, Pa., ALR
BURNS, S. S (1919)....Missouri Theater Bldg., St. Louis, Mo., ALR
BURRITT, N. W. (1930)....30 Beechwood Road, Summit, N. J., ALR
BURTON, FRANK A. (1914),
Medico-Dental Bldg., San Diego, Cal., ALR
*BUSHMAN, L. B. (1927),
631 City Nat'l Bank Bldg., Omaha, Neb., OALR
BUTLER, MARGARET F.....1831 Chestnut St., Philadelphia, Pa., ALR

*Butler, Robert H. (1920),
112 W. Columbus Ave., Bellefontaine, O., OALR
Butt, Miriam M.....2045 Chestnut St., Philadelphia, Pa., Op.
Buvinger, Charles W. (1921),
50 Washington St., East Orange, N. J., OALR
Buzzard, Josiah F. (1931).....1110 13th Ave., Altoona, Pa., Op.
Byrnes, H. F.....67 Chestnut St., Springfield, Mass., OALR

C

Cady, D. W. (1930).....65 N. Madison Ave., Pasadena, Cal., Op.
Cahill, John Wm.....390 Main St., Worcester, Mass., OALR
Caldwell, Robert,
831 New Donaghey Bldg., Little Rock, Ark., OALR
Calhoun, F. Phinizy (1911), 131 Forrest Ave., Atlanta, Ga., Op.
Calhoun, Jas. G.....Metropolitan Bldg., St. Louis, Mo., Op.
Callfas, W. F.....1620 Med. Arts Bldg., Omaha, Neb., ALR
Cameron, Wm. G...Rhodes Med. Arts Bldg., Tacoma, Wash., OALR
*Camp, W. E. (1920), Medical Arts Bldg., Minneapolis, Minn., OALR
Campbell, A. A. (1930).....86 Bloor St. W., Toronto, Ont., ALR
Campbell, C. H., Columbus State Bk. Bldg., Columbus, Neb., OALR
Campbell, D. A.....David Whitney Bldg., Detroit, Mich., OALR
Campbell, Don M. (1908),
David Whitney Bldg., Detroit, Mich., OALR
Campbell, M. D. (1927).....269 Rowena St., Detroit, Mich., OALR
Campbell, Robert A., 520 LaSalle Bldg., Minneapolis, Minn., OALR
Campbell, W. K.....96 Third Ave., Long Branch, N. J., OALR
Capron, F. P.....118 Angell St., Providence, R. I., OALR
Cardwell, Edgar P. (1929).....15 Fulton St., Newark, N. J., ALR
Carleton, Ralph (1914)....10 Temple St., Springfield, Mass., Op.
*Carmack, J. W. (1919),
Hume-Mansur Bldg., Indianapolis, Ind., ALR
*Carmody, T. E. (1904).....Metropolitan Bldg., Denver, Colo., ALR
Carmody, Thomas J. (1926),
408 The Temple Bldg., Danville, Ill., OALR
Carncross, Helen (1920),
58 E. Washington St., Chicago, Ill., Op.
Carpenter, Chapin (1930),
1930 Chestnut St., Philadelphia, Pa., Op.
Carpenter, E. R.....3805 Bowser Ave., Dallas, Tex., OALR
Carpenter, E. W. (1920).....E. North St., Greenville, S. C., OALR
Carr, Geo. W. (1909)....54 S. Franklin St., Wilkes-Barre, Pa., OALR
Carroll, Geo. G.....5 N. Goodman St., Rochester, N. Y., OALR
Carruth, Howard Ernest (1928)
Medical Arts Bldg., Portland, Ore., OALR
*Carson, W. E.....Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
Carter, E. D. (1930).....Central Bldg., Wichita, Kan., ALR
Carter, John M. (1929),
David Whitney Bldg., Detroit, Mich., ALR
Carter, W. W. (1896), 2 E. 54th St., New York City, N. Y., ALR

CARVILL, MAUD (1914)
82 Commonwealth Ave., Boston, Mass., Op.
CARY, ED. F. (1930)..... 171 W. 57th St., New York, N. Y., ALR
*CARY, E. H.....Medical Arts Bldg., Dallas, Tex., OALR
CARY, G. C.....Canon Block, Grand Junction, Colo., OALR
*CASSADY, J. VERNAL (1928)
654 Associates Bldg., South Bend, Ind., OALR
CASSIDY, WALDRON A. (1931),
Medical Arts Bldg., Omaha, Neb., ALR
CASTROVIEJO, ROMON (1931),
231 W. Washington St., Chicago, Ill., Op.
CAVANAUGH, J. A.....1712-30 N. Michigan Blvd., Chicago, Ill., ALR
CAVE, LINUS S. (1929).....Physicians' Bldg., Syracuse, N. Y., ALR
*CHADWICK, IRA B. (1920)..Terminal Bldg., Coffeyville, Kan., Op.
*CHAMBERLAIN, WEBB P. (1931),
7405 Detroit Ave., Cleveland, O., Op.
CHAFFERS, WM. H. (1931).....190 Bates St., Lewiston, Me., ALR
CHANDLER, G. E. (1930).....Huntington Bldg., Miami, Fla., ALR
CHAPMAN, S. JEFFERSON (1926),
402 Burns Bldg., Colorado Springs, Colo., ALR
*CHAPMAN, V. A.....312 E. Wisconsin Ave., Milwaukee, Wis., OALR
And 1275 East Green St., Pasadena, Cal.
*CHAPPELL, R. S. (1925),
Traction Terminal Bldg., Indianapolis, Ind., ALR
*CHARLES, JOS. W.....3720 Washington Ave., St. Louis, Mo., Op.
CHARLTON, C. COULTER,
124 S. Illinois Ave., Atlantic City, N. J., ALR
CHASE, E. FRANK (1918).....1008 Cobb Bldg., Seattle, Wash., OALR
CHASE, S. B. (1925).....Carver Bldg., Ft. Dodge, Ia., OALR
*CHASE, W. D. (1924).....230 E. Broad St., Bethlehem, Pa., ALR
CHATTIN, J. FRANKLIN.....671 Broad St., Newark, N. J., Op.
CHENERY, W. E.....377 Commonwealth Ave., Boston, Mass., ALR
CHENEY, JAMES W.....702 Schweiter Bldg., Wichita, Kan., OALR
CHILDERS, ROBT. J. (1929).....604 Park Ave., Plainfield, N. J., ALR
*CHILDS, H. A.....Eagle Blk., Creston, Ia., OALR
*CHIPMAN, L. D. V. (1925),
42 Coburg St., St. John, N. B., Canada, OALR
CHRISTOPH, CARL H. (1928),
30 N. Michigan Ave., Chicago, Ill., OALR
*CLARK, CECIL PRATT (1931),
Hume-Mansur Bldg., Indianapolis, Ind., Op.
CLARK, H. S. (1916), Medical Arts Bldg., Minneapolis, Minn., OALR
*CLARK, I. G. (1920).....188 E. State St., Columbus, O., OALR
CLARK, J. SHELDON (1914), 509 State Bk. Bldg., Freeport, Ill., OALR
CLARK, L. H. (1915).....337 Monroe Ave., Rochester, N. Y., OALR
CLAY, G. E. (1917).....Medical Arts Bldg., Atlanta, Ga., Op.
CLEFF, OSCAR (1926).....55 E. Washington St., Chicago, Ill., OALR
CLEMENT, C. C.....30 N. Michigan Blvd., Chicago, Ill., OALR
CLEMENT, L. O. (1930).....Guardian Bldg., Salem, Ore., OALR
CLERF, LOUIS HENRY (1923), 128 S. 10th St., Philadelphia, Pa., ALR

CLEVENGER, W. F. (1905),
Hume-Mansur Bldg., Indianapolis, Ind., ALR
CLIFFORD, T. P. (1927)
1802 David Whitney Bldg., Detroit, Mich., OALR
CLOTHIER, JOSEPH.....Kane Bldg., Pocatello, Idaho, OALR
*COATES, GEO. M. (1922).....1721 Pine St., Philadelphia, Pa., ALR
COBB, EDWIN (1912)
Masonic Temple Bldg., Marshalltown, Ia., OALR
*CODD, A. N. (1929)
Paulsen Medical and Dental Bldg., Spokane, Wash., ALR
CODY, CLAUDE CARR (1928)
1304 Walker Ave., Houston, Tex., ALR
COFFELT, THEODORE A., 1240 Benton Ave., Springfield, Mo., OALR
COGAN, J. E.....707 Rose Bldg., Cleveland, O., OALR
COHEN, HERMAN B. (1927)
"The Lenox," Spruce and 13th Sts., Philadelphia, Pa., ALR
*COHEN, MARTIN.....1 West 85th St., New York, N. Y., Op.
COHEN, SAMUEL (1920).....2009 Pine St., Philadelphia, Pa., ALR
COHN, FELIX.....47 W. 87th St., New York, N. Y., ALR
*COHOE, DON A. (1930)
13535 Woodward Ave., Highland Park, Detroit, Mich., ALR
COLBERT, R. M. (1929).....57 West 57th St., New York, N. Y., ALR
*COLLIER, GEORGE B. (1929)
1317 Whitney Bldg., New Orleans, La., ALR
COLLINS, E. W.....508 Majestic Bldg., Denver, Colo., ALR
COLLINS, J. D. (1914).....187 Main St., Northampton, Mass., ALR
COLVER, B. N. (1913).....Sanitarium 1, Battle Creek, Mich., ALR
COMEE, W. C. (1922).....Northern Bldg., Green Bay, Wis., OALR
CONE, ALFRED J. (1929)
Barnes Hospital, 600 S. Kingshighway, St. Louis, Mo., ALR
CONLON, F. A.....301 Essex St., Lawrence, Mass., OALR
CONNOR, CHAS. E. (1920).....Miller Clinic, St. Paul, Minn., ALR
*CONNOR, RAY (1905).....1410 Stroh Bldg., Detroit, Mich., OALR
CONSTANS, GEORGE M. (1929)
Quain and Ramstad Clinic, Bismarck, N. D., Op.
CONSTANTINOPLE, P. S. (1929)
1801 Eye St., N. W., Washington, D. C., ALR
CONWAY, J. M. (1930).....Jenkins Arcade, Pittsburgh, Pa., ALR
*COOK, W. ALBERT (1924).....Medical Arts Bldg., Tulsa, Okla., OALR
COOPER, C. E.....652 Metropolitan Bldg., Denver, Colo., ALR
COPPS, LYMAN ALDEN (1923)
Marshfield Clinic, Marshfield, Wis., OALR
*CORBETT, J. J. (1925).....520 Beacon St., Boston, Mass., OALR
*CORSER, J. B.....345 Wyoming Ave., Scranton, Pa., Op.
*CORTOPASSI, A. J. (1930)
Second Nat'l Bank Bldg., Saginaw, Mich., ALR
CORYA, H. W. (1930).....30 E. 40th St., New York, N. Y., ALR
*COSTEN, J. B. (1925), Beaumont Medical Bldg., St. Louis, Mo., ALR
COTT, C. C.....104 Dorchester Road, Buffalo, N. Y., ALR

COTT, D. N. (1924)
25 Central Ave., St. George, Staten Isl., New York, ALR
COULTER, CHAS. F.
404 First Nat'l Bank Bldg., Great Falls, Mont., OALR
*COWAN, ALFRED (1928), 1930 Chestnut St., Philadelphia, Pa., Op.
*COWLEY, ROBERT HENRY (1926).....Berea, Ky., OALR
COWPER, H. W.....543 Franklin St., Buffalo, N. Y., Op.
CRAFT, KENNETH L. (1920)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
CRANE, J. WELLINGTON (1921)
128 Third St., Trenton, N. J., OALR
CRANE, W. R. (1929).....756 S. Broadway, Los Angeles, Cal., ALR
CREIGHTON, WM. J. (1910), 258 S. 18th St., Philadelphia, Pa., Op.
*CRISP, WM. H. (1913).....Metropolitan Bldg., Denver, Colo., Op.
CROCKETT, R. L.....302 Main St., Oneida, N. Y., Op.
CROISSANT, CHAS. A. (1931)..390 Main St., Worcester, Mass., ALR
CROMAN, JOS. M., JR. (1924)
Washington Apts., Mount Clemens, Mich., OALR
CROSS, A. E. (1914).....390 Main St., Worcester, Mass., Op.
CROSS, G. H. (1919).....525 Welsh St., Chester, Pa., Op.
CROSSLEY, E. R.....30 N. Michigan Blvd., Chicago, Ill., OALR
CRUME, JOHN J.....Amarillo Bldg., Amarillo, Texas, ALR
CRUMRINE, NORMAN ROSS (1928), 595 Third St., Beaver, Pa., OALR
CULBERTSON, L. R. (1898).....Masonic Temple, Zanesville, O., OALR
*CULLOM, M. M. (1910)
1119 Bennie-Dillon Bldg., Nashville, Tenn., OALR
CUMMINGS, GEO. O. (1930).....47 Deering St., Portland, Me., ALR
CUNNING, DANIEL S. (1928)
145 E. 54th St., New York City, N. Y., OALR
CUNNINGHAM, ALLAN R. (1924)
260 Barrington St., Halifax, N. S., OALR
CURDY, R. J.....1102 Grand Ave., Kansas City, Mo., Op.
CURRAN, E. J. (1919)
1805 Federal Reserve Bank Bldg., Kansas City, Mo., Op.
CURRY, GLENDON E. (1912)
Westinghouse Bldg., Pittsburgh, Pa., Op.
*CURTIN, EUGENE A. (1923), 703 Connell Bldg., Scranton, Pa., OALR
*CUSHMAN, BEULAH (1924)
25 E. Washington St., Chicago, Ill., Op.
CUTINO, R. M. (1930).....165 Clinton St., Brooklyn, N. Y., Op.
CUTLER, FRANKLIN E.....R. D. No. 2, Cortland, N. Y., ALR

D

DABNEY, S. G.....Starks Bldg., Louisville, Ky., OALR
DABNEY, VIRGINIUS (1913)
1633 Connecticut Ave., Washington, D. C., ALR
DAILEY, GILBERT L.....618 N. 3rd St., Harrisburg, Pa., OALR
DAILY, LOUIS (1926)....1117 Med. Arts Bldg., Houston, Tex., OALR
DAILY, RAY K. (1926), 1117 Med. Arts Bldg., Houston, Tex., OALR

DANCY, ALEXANDER B.
First Nat'l Bank Bldg., Jackson, Tenn., OALR
DANIELS, R. L. ..... Box 685, New Bern, N. C., OALR
DARMER, GEO. A. (1921) ..... 401 Terminal Bldg., Aurora, Ill., OALR
DARROW, C. H. (1928), 110 Metropolitan Bldg., Denver, Colo., ALR
DAVIDSON, H. P. (1924)
1424-30 N. Michigan Ave., Chicago, Ill., Op.
DAVIDSON, MORRIS (1929), 210 W. 70th St., New York, N. Y., Op.
DAVIES, H. F. (1930), 1835 Eye St., N.W., Washington, D. C., ALR
DAVIES, J. S. (1927) ..... Medical Arts Bldg., Tacoma, Wash., OALR
DAVIES, T. A. (1913) ..... 578 Sherborne St., Toronto, Can., OALR
DAVIS, A. EDWARD ..... 40 E. 61st St., New York City, N. Y., Op.
DAVIS, CHARLES L. (1928)
1305 Medico-Dental Bldg., San Diego, Cal., ALR
DAVIS, F. A. (1927), 224 W. Washington Ave., Madison, Wis., OALR
DAVIS, G. E. ..... 1060 Fifth Ave., New York City, N. Y., OALR
DAVIS, HARRY B. (1920) ..... Bryant Bldg., Kansas City, Mo., OALR
*DAVIS, J. CALVIN (1928) ..... 425 Aquila Court, Omaha, Neb., OALR
DAVIS, J. E. ..... Kress Bldg., McAlester, Okla., OALR
DAVIS, J. L. ..... 135 S. 18th St., Philadelphia, Pa., ALR
DAVIS, RALPH F. (1920) ..... Mayer Bldg., Portland, Ore., OALR
DAVIS, THOMAS C. (1931)
3128 N. Broad St., Philadelphia, Pa., ALR
DAVIS, THOMAS W.
210 O'Hanlon Bldg., Winston-Salem, N. C., OALR
*DAVIS, WM. C. (1930) ..... 327 E. State St., Columbus, O., Op.
DAVIS, W. THORNWALL (1919)
927 Farragut Sq., Washington, D. C., Op.
*DAY, KENNETH M. (1930)
121 University Place, Pittsburgh, Pa., ALR
DAYTON, G. O. ..... 1401 S. Hope St., Los Angeles, Cal., OALR
DEAL, JOHN FRANCIS (1928)
First Nat'l Bank Bldg., Springfield, Ill., OALR
DEAN, ALFRED, 308 Medical Arts Bldg., Grand Rapids, Mich., Op.
DEAN, ARTHUR C. (1929)
Hot Springs Clinic, Hot Springs, S. D., ALR
DEAN, F. W. (1904) ..... 532 First Ave., Council Bluffs, Ia., OALR
*DEAN, L. W.
Medical School, Washington University, St. Louis, Mo., ALR
*DEAN, WALTER (1917) ..... 300 Francis Bldg., Louisville, Ky., OALR
*DEARMIN, ROBERT M. (1931)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
DEBOE, M. P. ..... First Nat'l Bank Bldg., Miami, Fla., OALR
DECHERD, H. B. ..... 1515 Medical Arts Bldg., Dallas, Tex., OALR
DECHTER, M. ARCHIBALD (1930)
Professional Bldg., Westfield, Mass., ALR
DECKER, J. C. (1918) ..... Frances Bldg., Sioux City, Ia., OALR
DECOSTE, STEPHEN H. (1931)
1179 Dean St., Brooklyn, N. Y., ALR
DEFNET, WM. A. (1921) .. 28 Adams Ave. West, Detroit, Mich., ALR

DEICHLER, LYN W.....1930 Chestnut St., Philadelphia, Pa., OALR
DELANEY, JAMES H. (1931).....138 West 9th St., Erie, Pa., Op.
DELPH, JOHN FRANCIS (1928)
104 S. Michigan Blvd., Chicago, Ill., ALR
DENISON, WARD C. (1931), 20 E. 53rd St., New York, N. Y., ALR
DENMAN, IRA O. (1915).....330 Richardson Bldg., Toledo, O., OALR
DENNIS, EDWARD P. (1921).....309 W. 10th St., Erie, Pa., OALR
DENNIS, F. L. (1906).....3811 Hawk St., San Diego, Cal., ALR
DENNIS, GEO. J. (1910)....25 E. Washington St., Chicago, Ill., ALR
DEPPING, C. W. (1930).....17 W. 54th St., New York, N. Y., ALR
DERBY, GEO. S.....5 Bay State Rd., Boston, Mass., Op.
DETLING, FRANK E.....500 S. Lucas Ave., Los Angeles, Cal., OALR
*DEWEESE, CLARENCE....201 West Second St., Lexington, Ky., OALR
DEWEY, C. H. (1912), Maj. M. C., U. S. Army,
U. S. Army Hosp., Ft. Benj. Harrison, Indianapolis, Ind., OALR
*DEWITT, EDWARD NICHOLAS (1928)
836 Myrtle Ave., Bridgeport, Conn., Op.A
DICKSON, JAMES C. (1931)
Medical Arts Bldg., Houston, Texas, ALR
DICKSON, JOHN FORREST (1928)
956 Shattuck Ave., Berkeley, Cal., OALR
*DILLON, IRA H. (1930)
A. T. & S. F. Ry. Hospital, Topeka, Kan., Op.
DINTENFASS, HENRY.....1305 Spruce St., Philadelphia, Pa., ALR
DIXON, FRED W. (1929).....Rose Bldg., Cleveland, O., ALR
DIXON, G. S.....1150 Fifth Ave., New York, N. Y., R
DIXON, OTTO JASON (1928)
Professional Building, Kansas City, Mo., ALR
DLUGASCH, LOUIS (1929)....110 E. 36th St., New York, N. Y., ALR
DOANE, L. L. (1905).....357 N. Main St., Butler, Pa., OALR
DOBBS, O. R. (1930).....2435 N. Grand Blvd., St. Louis, Mo., ALR
DODD, OSCAR (1903).....636 Church St., Evanston, Ill., Op.
DOHERTY, WM. B. (1927)
150 W. 55th St., New York, N. Y., Op.
DOLMAN, PERCIVAL (1919), 490 Post St., San Francisco, Cal., Op.
DONNELL, N. R. (1917)....Metropolitan Bldg., St. Louis, Mo., Op.
DONOHER, WM. D.....Boston Bldg., Salt Lake City, Utah, OALR
DONOHOE, ED. C. (1931)....Professional Bldg., Glendale, Cal., ALR
DONOVAN, JNO. A. (1902)....507 Phoenix Bldg., Butte, Mont., OALR
DORENTE, D. R.....First Nat'l Bank Bldg., Fort Smith, Ark., OALR
DORGE, RICHARD IRVING (1922)
2319 Sixth St., South, Minneapolis, Minn., OALR
DOUGHERTY, CLIFFORD L. (1931)
104 S. Michigan Ave., Chicago, Ill., ALR
DOUGLAS, FREDERICK ALEXANDER (1922)
303 State Bank Bldg., La Crosse, Wis., OALR
DOW, FRANK E. (1914).....4 West St., Northampton, Mass., Op.
DOW, JULIAN NEAL (1931)..727 W. 7th St., Los Angeles, Cal., Op.
DOWLING, J. IVIMEY.....116 Washington Ave., Albany, N. Y., OALR
DOWLING, J. T. (1915).....1115 Terry, Seattle, Wash., OALR

*Duane, J. F. (1916)..... Jefferson Bldg., Peoria, Ill., OALR
Duke, Roncie R. (1931)...Citizens' Bank Bldg., Tampa, Fla., ALR
*Dulaney, Octavus (1917)..811 Brown Bldg., Louisville, Ky., ALR
Duncan, R. A. (1929)...339 Amarillo Bldg., Amarillo, Texas, ALR
Dunlap, Lawrence G. (1920)
101 Main St., Anaconda, Mont., OALR
Dunn, Murison..... Richmond, Ky., OALR
Dunn, O. B..... 4th and Railroad Sts., Ironton, O., OALR
Dunnington, J. H. (1924)
30 W. 59th St., New York City, N. Y., Op.
Dunphy, Edwin B. (1931)
520 Commonwealth Ave., Boston, Mass., Op.
Duntley, G. S. (1913)..... 103 So. Side Sq., Macomb, Ill., OALR
Durr, S. A. (1927)
1304 Medico-Dental Bldg., San Diego, Cal., Op.
Dutrow, H. V. (1914)..1040 Fidelity Med. Bldg., Dayton, O., OALR
Dyer, B. G. (1929)..... Ames, Iowa, ALR
Dyer, C. P. (1920)..... 408 Humboldt Bldg., St. Louis, Mo., Op.
Dyer, Wallace C. (1929)
Central Union Bank Bldg., Evansville, Ind., ALR

E

*Eagleton, W. P. (1930)..... 15 Lombardy St., Newark, N. J., ALR
Earel, A. M. (1912)..... Willdon Bldg., Hoopeston, Ill., OALR
Earley, James Hugh (1928)
1801 Eye St., N. W., Washington, D. C., ALR
Earnest, Clarence E. (1931)..Corwin Hospital, Pueblo, Colo., ALR
Easton, J. C. (1904), First Nat'l Bank Bldg., Springfield, O., OALR
Ebeling, Wm. B. (1930)..... 54 Greene Ave., Brooklyn, N. Y., Op.
Eber, Carl Theodore (1928)
1006 Carleton Bldg., St. Louis, Mo., Op.
Ebert, Edwin C. (1929)
Naval Dispensary, Washington, D. C., ALR
Edgerton, Ambrose Earl (1931)
525 N. Rexford Drive, Beverly Hills, Cal., Op.
Edson, R. A..... 957 Delaware Ave., Buffalo, N. Y., Op.
Edwards, C. J. (1913)
First National Bank Bldg., Vicksburg, Miss., OALR
Edwards, Slocomb R. (1918)
Christman Bldg., South Bend, Ind., OALR
*Egan, Burton Wright (1928)
408 North St., Logansport, Ind., OALR
*Eimer, Charles Eugene (1928)
316 Lister Bldg., St. Louis, Mo., ALR
Ellett, E. C. (1898)..... Exchange Bldg., Memphis, Tenn., Op.
*Ellis, Bert E. (1931)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
Ellis, C. Ward (1928)..... 215 N. Walnut St., Lansing, Mich., ALR
Ellis, Z. H. (1927)..... 58 E. 65th St., New York, N. Y., Op.

ELWOOD, CALVIN R. (1922)....Spies Bldg., Menominee, Mich., OALR
EMBERY, FRANK (1927)
1532 Overington St., Philadelphia, Pa., ALR
EMERSON, LINN (1906).....Metropolitan Bldg., Orange, N. J., OALR
ENGLE, H. P.....527 West First Ave., Newton, Ia., OALR
*EQUEN, MURDOCK S.....Medical Arts Bldg., Atlanta, Ga., ALR
ERSNER, MATTHEW S.....1915 Spruce St., Philadelphia, Pa., ALR
ESTABROOK, CHAS. T.....857 Pleasant St., Worcester, Mass., OALR
*EUBANK, AMBROSE E. (1928)
636 Argyle Bldg., Kansas City, Mo., OALR
EVANS, J. N. (1925)....23 Schermerhorn St., Brooklyn, N. Y., Op.
*EZELL, H. C. (1926).....228 Doctors Bldg., Nashville, Tenn., OALR

F

*FACKLER, C. L. (1930).....11 E. Market St., York, Pa., ALR
FAIRBAIRN, JOHN F.....925 Delaware Ave., Buffalo, N. Y., ALR
FAIRCHILD, NORA M. (1922)..Med. Arts Bldg., Omaha, Neb., Op.
FAIRING, JOHN W. (1924)
206 Tribune Review Bldg., Greensburg, Pa., OALR
FAITH, THOMAS.....31 N. State St., Chicago, Ill., OALR
FALLOWS, HOWARD D. (1916)
Park Hospital Clinic, Mason City, Ia., OALR
*FARMER, A. G. (1919).....Fidelity Medical Bldg., Dayton, O., OALR
FARNSLER, H. HERSHEY.....1438 Market St., Harrisburg, Pa., OALR
FARNSWORTH, M. A. (1917)
Chamberlain Sanitarium and Hospital, Chamberlain, S. D., OALR
FARRINGTON, P. M. (1904)
Physicians and Surgeons Bldg., Memphis, Tenn., ALR
FARRIOR, J. BROWN (1928)
605 Citizens Bank Bldg., Tampa, Fla., OALR
FAWCETT, I. (1913).....75 12th St., Wheeling, W. Va., OALR
FEASTER, HENRY J. (1931).....461 77th St., Brooklyn, N. Y., ALR
*FENTON, RALPH A. (1921)
806 Medical Arts Bldg., Portland, Ore., OALR
FERGUSON, E. S. (1906)
902 Medical Arts Bldg., Oklahoma City, Okla., OALR
FERGUSON, R. J. (1927)....59 College St., New Haven, Conn., OALR
FERMAN, JOHN W. (1921).....Stahlman Bldg., Charleroi, Pa., OALR
FERNLUND, GEORGE BYRON (1928)
8555 118th St., Richmond Hill, New York, ALR
FERRIS, DAVID P. (1928), 1000 Carleton Bldg., St. Louis, Mo., ALR
FEWELL, A. G. (1931).....1924 Pine St., Philadelphia, Pa., Op.
*FIELDING, F. G. (1925).....70 Ridge St., Glen Falls, N. Y., ALR
FIGI, F. A. (1927).....Mayo Clinic, Rochester, Minn., LR
FINDLAY, E. K. (1930).....30 N. Michigan Ave., Chicago, Ill., Op.
*FINEBERG, MAXWELL (1930)
3858 Westminster Place, St. Louis, Mo., ALR
*FINK, WALTER H. (1927)
Medical Arts Bldg., Minneapolis, Minn., OALR

*Finnoff, W. C. (1916).....Republic Bldg., Denver, Colo., Op.
Fischbach, V. W. (1931)
2600 Union Central Bldg., Cincinnati, O., ALR
Fischer, N. Arthur (1925)
623 Union Trust Bldg., Pittsburgh, Pa., ALR
Fischer, V. B.....203 Mercantile Bank Bldg., Boulder, Colo., OALR
Fisher, Carl.....1930 Wilshire Blvd., Los Angeles, Cal., Op.
*Fisher, James A. (1925)
Jersey Central Bldg., Asbury Park, N. J., OALR
Fisher, W. A. (1896), 231 W. Washington St., Chicago, Ill., OALR
Fiske, Geo. F.....25 E. Washington St., Chicago, Ill., OALR
Fitzgerald, J. P. (1924), 30 N. Michigan Blvd., Chicago, Ill., Op.
Flagg, J. D. (1900).....158 Anderson Place, Buffalo, N. Y., Op.A.
Flatley, T. J. (1914).....Reliance Bldg., Moline, Ill., OALR
Flock, Herman F. W. (1931)
416 Pine St., Williamsport, Pa., ALR
Floyd, T. W. (1918).....811 Peoria Life Bldg., Peoria, Ill., OALR
Flynn, Chas. T. (1927)
41 Trumbull St., New Haven, Conn., ALR
*Flynn, James A. (1924)
1511 Rhode Island Ave., N. W., Washington, D. C., ALR
Fogarty, Chas. W. (1920), 372 St. Peter St., St. Paul, Minn., OALR
Foley, J. W. (1915).....Majestic Bldg., Denver, Colo., OALR
*Folk, Max Lyon (1928), 55 E. Washington St., Chicago, Ill., OALR
Folken, Frank Garrett (1928)
306 Albert Lea State Bank Bldg., Albert Lea, Minn., OALR
*Folsom, W. H. (1927)....11 N. Main St., Fond du Lac, Wis., OALR
*Forbes, Sherman B. (1921)
409 Citizens Bank Bldg., Tampa, Fla., OALR
*Ford, Hanby Lewis (1928)
401 Robeson Bldg., Champaign, Ill., OALR
*Forgrave, L. Robert (1922), 204 Logan Bldg., St. Joseph, Mo., OALR
Forsythe, S. T. (1919).....Rose Bldg., Cleveland, O., OALR
Fort, A. G.....Doctors Bldg., Atlanta, Ga., OALR
Foster, J. H.....1304 Walker Ave., Houston, Texas, ALR
*Fouts, John D. (1920).....Fidelity Bldg., Dayton, O., OALR
*Fowler, Earl B. (1925)..55 E. Washington St., Chicago, Ill., Op.
Fowler, Robt. H. (1929)..153 E. 62nd St., New York, N. Y., ALR
Fowler, S. R. (1910).....University Blk., Syracuse, N. Y., ALR
Fowler, Wm. (1925).....Maccabee Bldg., Detroit, Mich., OALR
Fowlkes, John W.....30 W. 59th St., New York, N. Y., ALR
Fox, C. H. (1920).....5 W. 21st St., Kearney, Neb., OALR
Fox, Noah (1926).....841 E. 63rd St., Chicago, Ill., ALR
Fox, Ralph D. (1916).....Unity Bldg., Bloomington, Ill., OALR
Franchere, Fred'k E. (1920)
Toy Bank Bldg., Sioux City, Ia., OALR
Frank, Ira (1908).....104 S. Michigan Blvd., Chicago, Ill., ALR
Frankel, Edward, Jr. (1928)
152 W. 58th St., New York City, N. Y., ALR

*Fraser, R. H. (1925)
Old Merchants Tower, Battle Creek, Mich., ALR
Fraser, Robt. C. (1929), 940 Military St., Port Huron, Mich., ALR
Fraser, W. G. (1927)....Medical Arts Bldg., Ottawa, Canada, OALR
Freedman, L. M. (1920)
475 Commonwealth Ave., Boston, Mass., ALR
*Freimuth, L. E. (1927)
University Club Bldg., St. Louis, Mo., ALR
French, R. F. (1913)....Masonic Temple, Marshalltown, Ia., OALR
Friedenwald, Harry.....1212 Eutaw Pl., Baltimore, Md., Op.A.
*Friedenwald, J. S. (1925)....1212 Eutaw Pl., Baltimore, Md., Op.
Fringer, W. R.....William Brown Bldg., Rockford, Ill., Op.
*Frost, Albert Dalbey (1928)
681 E. Broad St., Columbus, O., Op.
Frothingham, Edward (1928)
8644 94th St., Woodhaven, N. Y., OALR
*Fry, W. E. (1931).....1912 Spruce St., Philadelphia, Pa., Op.
*Fuchs, Valentine Henry (1928)
220 Chaille Bldg., New Orleans, La., ALR
Fulkerson, C. B. (1914)
Room 403, 107 W. Michigan Ave., Kalamazoo, Mich., OALR
Fullenwider, C. M.....Barnes Bldg., Muskogee, Okla., OALR
Fuller, Harry B. (1925)....2219 E. 70th Place, Chicago, Ill., OALR
Fuller, John Andre (1928)
First National Bank Bldg., Reno, Nev., OALR
Fuller, T. E. (1915)
Texarkana Nat'l Bank Bldg., Texarkana, Tex., OALR
Fulton, John F. (1920)..833 Lowry Bldg., St. Paul, Minn., OALR
Furgason, A. P.....Richards Blk., Lincoln, Neb., OALR
*Furstenberg, A. C. (1925)
First National Bank Bldg., Ann Arbor, Mich., ALR
Fust, John H. E. (1922).....221 W. 9th St., Erie, Pa., OALR

G

Gabriel, C. K. (1930)
Western Catholic Union Bldg., Quincy, Ill., ALR
Gadbois, Arthur E.(1921), 421 Norfolk Ave., Norfolk, Neb., OALR
*Gailey, Watson W. (1923)
Griesheim Bldg., Bloomington, Ill., OALR
Gale, M. Jean (1919).....Republic Bldg., Denver, Colo., Op.
Gallagher, J. H. C. (1927)..105 Center St., Chicopee, Mass., ALR
Gill, Arthur Ernest (1928)
U. S. Veterans' Hosp., Fort Bayard, N. M., OALR
*Gill, E. G. (1919).....Box 871, Roanoke, Va., ALR
*Gill, G. Bache (1923)
1624 Eye St., N. W., Washington, D. C., ALR
Gill, Wm. Davis (1929)
323 Medical Arts Bldg., San Antonio, Texas, OALR

GALLAHER, T. J.....California Bldg., Denver, Colo., ALR
GALLOWAY, T. C. (1927).....636 Church St., Evanston, Ill., ALR
*GAMBLE, R. C. (1929).....30 N. Michigan Ave., Chicago, Ill., Op.
GARBER, J. M.....14 N. Mulberry St., Mansfield, O., OALR
GARRAGHAN, EDW. F.....25 E. Washington St., Chicago, Ill., OALR
GARRETT, J. R. (1913).....Mt. Trust Bldg., Roanoke, Va., OALR
GATEWOOD, E. T. (1925), Professional Bldg., Richmond, Va., ALR
GATEWOOD, W. L.....330 Park Ave., New York City, N. Y., ALR
GAUDET, LUCIEN SIDNEY (1928)
405 Franklin St., Natchez, Miss., OALR
GEIGER, A. H. (1929).....554 Fullerton Parkway, Chicago, Ill., ALR
GEIGER, C. W. (1904).....Arcade Bldg., Kankakee, Ill., OALR
*GEORGE, HENRY W. (1919), 19 N. Union St., Middletown, Pa., Op.
GIBBY, HAROLD J. (1931).....27 Elm St., Worcester, Mass., ALR
*GIFFORD, S. R. (1919).....58 E. Washington St., Chicago, Ill., Op.
GILBERT, FRANK Y.....148 Park St., Portland, Me., OALR
GILLETT, WILBUR G.....230½ N. Main St., Wichita, Kan., Op.
*GILLETTE, D. F. (1925).....State Tower Bldg., Syracuse, N. Y., Op.
GILLILAND, O. S. (1930), Professional Bldg., Kansas City, Mo., ALR
GILLUM, J. R. (1920).....221 S. 6th St., Terre Haute, Ind., OALR
GIORDANO, ANTHONY J. (1931)
749 E. 183rd St., New York, N. Y., ALR
GIPNER, J. F. (1926).....53 S. Fitzhugh St., Rochester, N. Y., Op.
GITTENS, T. R. (1920).....401 Davidson Bldg., Sioux City, Ia., OALR
GLASFORD, S. T. (1924).....Farmers' Bank Bldg., Pekin, Ill., OALR
GLATT, MORRIS A.....3254 Lawrence Ave., Chicago, Ill., ALR
GLEASON, E. B.....2112 Pine St., Philadelphia, Pa., ALR
*GLICK, HARRY NAYLOR (1928)
3115 S. Grand Blvd., St. Louis, Mo., ALR
*GLOSSER, HERBERT H. (1912)
448 Franklin St., Buffalo, N. Y., Op.
GLOVER, S. P.....1118 Twelfth Ave., Altoona, Pa., OALR
GLOWACKI, BEN FRANCIS (1928)
770 Maccabee Bldg., Detroit, Mich., OALR
GOAD, R. R. (1931).....Laurel Bldg., Muscatine, Iowa, Op.
GOAR, EVERETT LOGAN (1926)
1300 Walker Ave., Houston, Tex., Op.
GODDARD, H. M.....1531 Spruce St., Philadelphia, Pa., ALR
GOLDBACK, L. J. (1911), 1012 N. Charles St., Baltimore, Md., Op.
GOLDENBURG, MICHAEL.....104 S. Michigan Ave., Chicago, Ill., Op.
GOLDING, JOSEPH E. (1925), 170 Hancock St., Brooklyn, N. Y., Op.
GOLDMAN, A. MILTON (1928)
121 E. 60th St., New York, N. Y., Op.
GOLDMAN, H. G.....57 W. 58th St., New York, N. Y., ALR
*GOLDSMITH, PERRY G.....Medical Arts Bldg., Toronto, Can., ALR
GOLDSTEIN, ISADORE (1929), 1060 Park Ave., New York, N. Y., Op.
GOLDSTEIN, M. A. (1896)
3858 Westminster Pl., St. Louis, Mo., ALR
GOOD, WM. M. (1929).....63 Center St., Waterbury, Conn., ALR

GOODMAN, THOMAS L...Medical Arts Bldg., Fort Worth, Tex., ALR
GOODE, ED A. (1929)....143-28 Sanford Ave., Flushing, N. Y., ALR
*GOODYEAR, H. M. (1924).....Doctors Bldg., Cincinnati, O., ALR
GORDON, CHAS. H. (1926).....46 Deering St., Portland, Me., ALR
GORDON, GLENN G. (1929)..1st Nat'l Bank Bldg., Baker, Ore., ALR
GORDON, J. S. (1919)..425 E. Wisconsin Ave., Milwaukee, Wis., OALR
GORRELL, T. J. H.
16th and Vincennes Ave., Chicago Heights, Ill., OALR
GOSNEY, C. W. (1920), Medical Arts Bldg., Los Angeles, Cal., Op.
GOTTLIEB, MARK J.....2 West 87th St., New York, N. Y., ALR
GOUGH, ROY H.....Medical Arts Bldg., Fort Worth, Texas, OALR
GOURLEY, G. F. (1916).....Sinclair Bldg., Steubenville, O., OALR
*GOUTERMAN, J. I.....1930 Chestnut St., Philadelphia, Pa., Op.
*GOUX, RAYMOND SALOT
545 David Whitney Bldg., Detroit, Mich., OALR
GOWEN, FRANCIS V. (1929)
904 E. Chelton Ave., Philadelphia, Pa., ALR
GOWEN, GEO. R. (1929)
306 Baker Bldg., Walla Walla, Wash., ALR
*GRACEY, GEO. F.....209 State St., Harrisburg, Pa., OALR
*GRADLE, H. S. (1911).....58 E. Washington St., Chicago, Ill., Op.
GRANGER, W. B. (1920)
Citizens Nat'l Bank Bldg., Emporia, Kan., OALR
GRANT, FRED E. (1912)
Kalamazoo Nat'l Bank Bldg., Kalamazoo, Mich., OALR
GRANT, H. W. (1930).....Hamm Bldg., St. Paul, Minn., Op.
GRANT, LEE E. (1919)..David Whitney Bldg., Detroit, Mich., OALR
*GRANT, P. T. (1919)
Medical Arts Bldg., Grand Rapids, Mich., OALR
GRAY, E. B. (1927)
909 Montgomery Bldg., Spartansburg, S. C., ALR
*GREEAR, J. N., JR. (1929)
1740 M St., N. W., Washington, D. C., Op.
GREEN, A. S.
The Green's Eye Hosp., 1801 Bush St., San Francisco, Cal., Op.
*GREEN, JOHN (1905), 3720 Washington Blvd., St. Louis, Mo., Op.
GREEN, L. D.
The Green's Eye Hosp., 1801 Bush St., San Francisco, Cal., Op.
GREENE, JOSEPH BERRY (1923)
Haywood Bldg., Asheville, N. C., ALR
GREENE, LAURENCE WHITRIDGE (1928)
Republic Bldg., Denver, Colo., ALR
GREENE, M. L. (1930), Missouri Theatre Bldg., St. Louis, Mo., Op.
GREENFIELD, SAMUEL D. (1927)
169 New York Ave., Brooklyn, N. Y., ALR
GREENFIELD, WILLIAM JOHN (1928)
50 Anderson St., Hackensack, N. J., ALR
GREENWOOD, ALLEN (1911)
82 Commonwealth Ave., Boston, Mass., Op.

GREENWOOD, G. J. (1930).....708 Church St., Evanston, Ill., ALR
GREEVER, B. L. (1920)
610 Rorabaugh-Wiley Bldg., Hutchinson, Kan., OALR
GREGG, JOHN B.
612 Security Nat'l Bank Bldg., Sioux Falls, S. D., OALR
GRESSER, ED B. (1929).....39 Fifth Ave., New York, N. Y., Op.
GRIFFEY, ED W. (1929), 1300 Walker Ave., Houston, Texas, ALR
GRIFFITH, H. M. (1930)
Johnstown Trust Bldg., Johnstown, Pa., Op.
GRIM, U. J.....30 N. Michigan Blvd., Chicago, Ill., OALR
GRISCOM, J. MILTON.....2213 Walnut St., Philadelphia, Pa., Op.
GROSS, JULIUS E. (1920)
8030 Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
GROSVENOR, L. N. (1907), 366 Dakota Ave., So. Huron, S. D., OALR
GROVE, W. E. (1914), 324 E. Wisconsin Ave., Milwaukee, Wis., OALR
GRUSHLOW, ISRAEL....271 Central Park W., New York, N. Y., ALR
GSELL, J. F. (1920).....911 Beacon Bldg., Wichita, Kan., OALR
GUERINOT, A. J. (1918)
604 Westinghouse Bldg., Pittsburgh, Pa., OALR
*GUGGENHEIM, L. K.....Carleton Bldg., St. Louis, Mo., ALR
GUNDELACH, C. A., 825 University Club Bldg., St. Louis, Mo., ALR
GUNN, M. L. (1929).....Smith Bldg., Harlan, Ky., ALR
GUTHRIE, A. L. (1910)....Osler Bldg., Oklahoma City, Okla., OALR
*GUTTMAN, M. REESE (1929)
185 N. Wabash Ave., Chicago, Ill., ALR
GUYTON, B. S. (1918)....512 Van Buren Ave., Oxford, Miss., OALR

H

HAAS, FREDERICK J. (1920)
Masonic Bldg., Leavenworth, Kan., OALR
HACKNEY, F. J. (1913)
630 Volunteer Bldg., Chattanooga, Tenn., OALR
HADEN, HENRY C.....1914 Travis St., Houston, Texas, Op.
HAESSLER, F. HERBERT (1929)
120 E. Wisconsin Ave., Milwaukee, Wis., Op.
HAGEMANN, JOHN A. (1925)
Highland Bldg., Pittsburgh, Pa., ALR
HAGLER, E. E. (1896)....401 E. Capitol Ave., Springfield, Ill., OALR
HAHN, WM. H. (1930).....15 Lombardy St., Newark, N. J., Op.
HALE, ARTHUR STANLEY (1928)
1609 Eaton Tower, Detroit, Mich., OALR
HALL, ALICE K. (1924).....25 E. Washington St., Chicago, Ill., ALR
HALL, EDWIN PERRY (1928)
State Tower Bldg., Syracuse, N. Y., ALR
HALL, G. C. (1907).....705 Brown Bldg., Louisville, Ky., OALR
HALLETT, G. DEWAYNE (1927)
274 W. 86th St., New York City, N. Y., Op.
HALSTED, FRED S.....738 Metropolitan Bldg., Denver, Colo., OALR

*HAMLIN, FRED EUGENE (1926)
Medical Arts Bldg., Roanoke, Va., ALR
*HAMPSEY, A. R. (1930).....May Bldg., Pittsburgh, Pa., ALR
HAMPTON, ROBT. R....804 Boston Bldg., Salt Lake City, Utah, ALR
HANCOCK, J. C. (1914), 135 Cambridge Pl., Brooklyn, N. Y., Op.
Also 2 Rector St., New York, N. Y.
HANDS, SIDNEY GILLOTT (1926)
201 West 2nd St., Davenport, Ia., ALR
HANEY, WM P. (1920)
1500 Medical Arts Bldg., Omaha, Neb., OALR
HANS, C. L. (1921).....Provident Bank Bldg., Cincinnati, O., OALR
*HANSEL, FRENCH KELLER (1926)
Beaumont Medical Bldg., St. Louis, Mo., ALR
HANSEN, ERLING W. (1920)
Medical Arts Bldg., Minneapolis, Minn., OALR
HANSON, WM. L. (1929)
First National Bank Bldg., East St. Louis, Ill., ALR
*HARALSON, CHAS. H. (1929)
816 Medical Arts Bldg., Tulsa, Okla., Op.
HARBERT, JOHN P. (1918)..135 N. Main St., Bellefontaine, O., OALR
*HARDESTY, J. F. (1920)
Missouri Theatre Bldg., St. Louis, Mo., Op.
HARDIE, G. C. (1930)....200 W. Michigan Ave., Jackson, Miss., ALR
HARDY, LE GRAND HAVEN (1928)
30 E. 40th St., New York City, N. Y., Op.
*HARDY, W. F. (1914).....Humboldt Bldg., St. Louis, Mo., Op.
*HARGITT, CHARLES A. (1926)
17 Schermerhorn St., Brooklyn, N. Y., Op.
*HARKNESS, GORDON F. (1911), Putnam Bldg., Davenport, Ia., OALR
*HARRELL, VOSS (1926).....Hofman Bldg., Detroit, Mich., ALR
HARRIS, C. M. (1910)
604 Johnstown Trust Bldg., Johnstown, Pa., OALR
HARRIS, ED W. (1930).....681 E. Broad St., Columbus, O., ALR
HARRIS, H. B.....Fidelity Med. Bldg., Dayton, O., OALR
HARRIS, J. HARLEY (1931)..Med. Arts Bldg., Memphis, Tenn., ALR
HARRIS, THOMAS J. (1929), 104 E. 40th St., New York, N. Y., ALR
HARRIS, W. C. (1900).....Groton Bldg., Cincinnati, O., ALR
HARRIS, WM. ROBIN (1923)
500 Lamar Life Bldg., Jackson, Miss., ALR
*HARRISON, FRED HAZARD (1928)
308 First Nat'l Bank Bldg., Connellsville, Pa., ALR
HARRISON, W. G.....Empire Bldg., Birmingham, Ala., OALR
HARTER, JOHN H. (1929)....320 Stimson Bldg., Seattle, Wash., ALR
HARTLEY, F. A.....302 E. High St., Springfield, O., OALR
HARTSHORNE, ISAAC (1926)
30 W. 59th St., New York City, N. Y., Op.
HARTZELL, S. M.
Dollar Savings and Trust Co., Youngstown, O., OALR

HARVEY, N. D.....112 Waterman St., Providence, R. I., OALR
HASELTINE, BURTON (1927)
122 S. Michigan Ave., Chicago, Ill., OALR
HASKELL, ALFRED W.....129 Park St., Portland, Me., Op.
HASKIN, W. H. (1913).....57 W. 57th St., New York, N. Y., ALR
HASPEL, M. DAVID (1911)
Maison Blanche Bldg., New Orleans, La., OALR
*HASTY, F. E. (1925)....Medical Arts Bldg., Nashville, Tenn., ALR
HATCH, C. B. (1908).....3 W. Church St., Newark, O., OALR
HAUER, A. M. (1916).....327 E. State St., Columbus, O., ALR
HAUGHEY, WILFRED H. (1911)
303 Post Bldg., Battle Creek, Mich., OALR
HAUPT, WM. C. (1931).....1235 Dean St., Brooklyn, N. Y., ALR
HAWLEY, C. W.....Medical Arts Bldg., Chicago, Ill., OALR
HAWMAN, ERLE G.....4051 N. Broad St., Philadelphia, Pa., Op.
*HAYDEN, AUSTIN A. (1910)
25 E. Washington St., Chicago, Ill., OALR
HAYS, HAROLD M. (1912)....64 E. 58th St., New York, N. Y., ALR
HAZLETT, L. R. (1913), Savings and Trust Bldg., Butler, Pa., OALR
HEALEY, JOHN F. (1924)..925 Delaware Ave., Buffalo, N. Y., ALR
HEARD, C. F. (1913).....813 Sassafras St., Erie, Pa., OALR
HEARD, MARY K.....128 19th Ave., North, St. Petersburg, Fla., Op.
HEATH, PARKER (1924), David Whitney Bldg., Detroit, Mich., Op.
HEBERT, ARTHUR W.....450 Sutter St., San Francisco, Cal., OALR
HECKEL, EDWARD B. (1902)
Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
HEDRICK, HAROLD B. (1929)
910 Rialto Bldg., Kansas City, Mo., ALR
HEEB, HARRY J.....204 Wisconsin Ave., Milwaukee, Wis., OALR
HEED, C. R.....1205 Spruce St., Philadelphia, Pa., Op.
HEETDERKS, D. R. (1930)
Medical Arts Bldg., Grand Rapids, Mich., ALR
HEEGIE, N. M. (1905).....Buckman Bldg., Jacksonville, Fla., OALR
HEINBERG, CHAS J. (1931).....Blount Bldg., Pensacola, Fla., ALR
HEITGER, J. D. (1905).....First Trust Bldg., Pasadena, Cal., OALR
*HELFRICH, ED D. (1930).....269 E. State St., Columbus, O., ALR
HEMPSTEAD, BERT E.....Mayo Clinic, Rochester, Minn., ALR
HENDERSON, L. E. (1930), 168 Sterling St., Watertown, N. Y., Op.
HENDRICKS, LOUIS J. (1931)
210 W. 12th St., Cincinnati, Ohio, Op.
HENNESSEY, WM. W.....333 Essex St., Salem, Mass., OALR
HENNING, CARL.....1835 Eye St., N. W., Washington, D. C., Op.
HENNINGER, L. L. (1913)..Professional Bldg., Pasadena, Cal., OALR
HENTON, G. E. (1918).....Morgan Bldg., Portland, Ore, OALR
HERRIMAN, L. L. (1913).....Physicians Bldg., Alamosa, Colo, OALR
HESS, WM. L. (1904).....La Jolla, Cal., OALR
HETRICK, L. E. (1923), 40 E. 61st St., New York City, N. Y., ALR
HETZEL, C. C. (1906).....Eccles Bldg., Ogden, Utah, OALR

HEWSON, WM. (1931).....1721 Pine St., Philadelphia, Pa., ALR
HICKEY, H. L. (1930).....Republic Bldg., Denver, Colo., ALR
HICKS, VONNIE MONROE (1926)
Professional Bldg., Raleigh, N. C., Op.
*HICKS, WM. J. (1930).....15 King St., Middletown, N. Y., ALR
HIGBEE, D. R. (1927).....3245 Fourth St., San Diego, Cal., ALR
HIGGINS, R. P. (1924).....20 Court St., Cortland, N. Y., OALR
*HIGGINS, S. G.....120 E. Wisconsin Ave., Milwaukee, Wis., OALR
HIGH, ISAAC B. (1931).....326 N. 5th St., Reading, Pa., ALR
*HILDING, ANDERSON C. (1931)..Mayo Clinic, Rochester, Minn., ALR
HILGARTNER, H. L., JR (1930)..Norwood Bldg., Austin, Tex., Op.
HILL, C. E. (1919).....160 Bloor St., W., Toronto, Can., Op.
HILL, EMORY (1916).....Professional Bldg., Richmond, Va., Op.
HILL, FREDERICK THAYER (1928)
Professional Bldg., Waterville, Me., ALR
HILL, HOWARD FOSTER (1928)
Professional Bldg., Waterville, Me., Op.
HINMAN, E. E. (1924)..146 Washington Ave., Albany, N. Y., ALR
HITZ, H. B.....425 E. Wisconsin Ave., Milwaukee, Wis., ALR
HOBART, CARL (19–9).....Metropolitan Bldg., St. Louis, Mo., Op.
HODGE, GEO. E. (1930).....Med. Arts Bldg., Montreal, Can., ALR
HODSON, B. F.....418 Security Bldg., Miami, Fla., OALR
HOFFMANN, JOHN N. (19–7).....309 Brant Bldg., Canton, O., ALR
HOFFMAN, L. G. (1927)....30 N. Michigan Ave., Chicago, Ill., OALR
HOFFMAN, WALTER F. (1920)
817 Summit Ave., Seattle, Wash., Op.
HOFLING, CHAS. A. (1929).....707 Race St., Cincinnati, O., Op.
*HOFMANN, W. P. (1925).....707 Kahl Bldg., Davenport, Ia., OALR
HOGAN, W. L. (1924).....750 Main St., Hartford, Conn., OALR
*HOGSHEAD, J. M.....602 Georgia Ave., Chattanooga, Tenn., OALR
HOGUE, D. W. (1903).....34 W. High St., Springfield, O., OALR
HOHF, JULIUS (1915).....Clinic Bldg., Yankton, S. D., OALR
HOLDSWORTH, FRANK
People's Savings Bank Bldg., Traverse City, Mich., OALR
HOLLENDER, ABRAHAM R. (1931)
30 N. Michigan Ave., Chicago, Ill., ALR
HOLLOWAY, THOS. B.....1819 Chestnut St., Philadelphia, Pa., Op.
HOLMES, BENJAMIN H. (1922), 435 Baker Blk., Racine, Wis., ALR
HOLT, E. E., JR .....723 Congress St., Portland, Me., Op.
HOLZER, WM. F. (1931)....36 Pleasant St., Worcester, Mass., Op.
*HOMPES, J. J.....702 Sharp Bldg., Lincoln, Neb., OALR
HOOPLE, GORDON D. (19–9), Med. Arts Bldg., Syracuse, N. Y., ALR
HOOSS, C. H. A. (1930)....3115 S. Grand Blvd., St. Louis, Mo., Op.
HOPKINS, G. H. (1930).....702 N. Main St., Pueblo, Colo., Op.
HOPKINS, W. E.....321 S. Lorraine Blvd., Los Angeles, Cal., OALR
HORN, W. L. (1929).....1040 Park Ave., New York, N. Y., ALR
HOTCHKISS, WALTER T. (1931)
1213 Lincoln Road, Miami Beach, Fla., ALR

*Hourn, George E. (1920)
511 University Club Bldg., St. Louis, Mo., ALR
Hoyde, A. G. (1916).....Board of Trade Bldg., Superior, Wis., OALR
Howard, C. N. (1919).....Warsaw Clinic, Warsaw, Ind., OALR
Howard, E. F. (1919)
First Nat'l Bank Bldg., Vicksburg, Miss., ALR
Howard, Edward L. (1926)
405 Medical Arts Bldg., Fort Worth, Tex., OALR
*Howard, Harvey J. (1928)
School of Med., Washington University, St. Louis, Mo., Op.
Howard, J. W.....Rialto Bldg., Kansas City, Mo., OALR
Howard, Lloyd G. (1923)
Council Bluffs Clinic, Council Bluffs, Iowa, OALR
Howard, Robt. Ed (1931)
2600 Union Central Bldg., Cincinnati, Ohio, ALR
Howard, W. H. (1923)
Medical Arts Bldg., Minneapolis, Minn., OALR
Howe, A. W. (1922).....Medical Arts Bldg., Tacoma, Wash., OALR
Howe, Spencer S. (1929)
419 Bellingham Nat'l Bank Bldg., Bellingham, Wash., ALR
Howell, Donald MacLean (1931)
1608 Eaton Tower, Detroit, Mich., ALR
Hubbard, Wm. B. (1931).....Dryden Bldg., Flint, Mich., ALR
Hughes, J. A. (1927).....Sterick Bldg., Memphis, Tenn., OALR
Hughes, L. W. (1925).....965 Broad St., Newark, N. J., OALR
Hughes, Ray Whitbeck (1928)
1501 David Whitney Bldg., Detroit, Mich., OALR
Hughes, T. E. (1922).....1000 W. Grace St., Richmond, Va., ALR
*Hughes, W. F., 401 Hume-Mansur Bldg., Indianapolis, Ind., Op.
Hughes, Wendell L. (1929)
20 E. 53rd St., New York, N. Y., Op.
Hume, John R. (1931)
Maison Blanche Bldg., New Orleans, La., ALR
Hunt, Westley M. (1929), 33 E. 68th St., New York, N. Y., ALR
Hunter, J. F. (1930).....536 Penn Ave., Turtle Creek, Pa., ALR
Hunter, R. J. (1930)....2011 Chestnut St., Philadelphia, Pa., ALR
*Hurd, L. M. (1910)....39 E. 50th St., New York City, N. Y., ALR
Hurff, J. W. (1919).....86 Washington St., Newark, N. J., OALR
Hurley, E. D. (1914).....419 Boylston St., Boston, Mass., Op.
Hurst, V. Reeves (1923), 107 E. Methvin St., Longview, Tex., OALR
Husik, D. N. (1921).....1930 Chestnut St., Philadelphia, Pa., ALR
Hussey, E. J.....245 Maple St., Holyoke, Mass., OALR
Hutchinson, Abbott Trask (1928)
30 W. 59th St., New York, N. Y., ALR
*Hutchison, K. O....1509 Sherbrooke St. W., Montreal, Can., ALR

I

*IGLAUER, S.....707 Race St., Cincinnati, O., ALR
IMPERATORI, C. J.....17 E. 38th St., New York, N. Y., ALR
INGERMAN, S. M. (1909)
418 Central Park, W., New York, N. Y., OALR
INGERSOLL, J. M. (1908).....Aladdin Bldg., Miami Beach, Fla., ALR
INGLIS, HARRY J.....43 Bay State Rd., Boston, Mass., ALR
INGRAM, L. C. (1915).....Orlando Clinic, Orlando, Fla., OALR
IRVIN, EDGAR H.....903 First Nat'l Bk. Bldg., El Paso, Texas, OALR
*IRVINE, A. RAY (1918)
1144 Roosevelt Bldg., Los Angeles, Cal., Op.
IRVINE, ROBT S. (1929).....490 Post St., San Francisco, Cal., ALR
IRWIN, V. J., SR (1909).....1634 Main St., Springfield, Mass., OALR
IRWIN, VINCENT J., JR. (1921)
1634 Main St., Springfield, Mass., OALR
*ISRAEL, NORMA E. (1919)
2001 Niels Esperon Bldg., Houston, Tex., OALR
ISRAEL, SIDNEY.....2001 Niels Esperon Bldg., Houston, Tex., ALR
IVINS, H. M. (1910)
815 Merchants Nat'l Bank Bldg., Cedar Rapids, Ia., OALR

J

JACKLE, ARTHUR F. (1917)..63 E. Fourth St., Dunkirk, N. Y., Op.
JACKSON, ATRAS EARL (1928)
Med. Arts Bldg., Fort Worth, Tex., Op.
JACKSON, CHEVALIER.....235 S. 15th St., Philadelphia, Pa., L
JACKSON, J. D. (1920).....232 West 8th St., Erie, Pa., OALR
*JACOBS, MAX W. (1916)
Missouri Theater Bldg., St. Louis, Mo., Op.
JACOBS, S. M.....1018 E. 163rd St., New York, N. Y., OALR
JACOBY, D. P. A. (1912).....106 Touro St., Newport, R. I., Op.AL
JAMES, WM. M. (1931)..640 S. Kingshighway, St. Louis, Mo., Op.
JAMESON, PATRICK C. (1931)
139 Montague St., Brooklyn, N. Y., Op.
*JAQUISH, CHARLES JOSEPH (1928)
904 Westinghouse Bldg., Pittsburgh, Pa., ALR
JARECKY, HERMAN.....168 W. 86th St., New York City, N. Y., OALR
*JARVIS, DEFOREST C. (1929).....Quarry Bank Bldg., Barre, Vt., ALR
JEAN, GEO. W.....7 East Valerio, Santa Barbara, Cal., Op.
*JENKINS, M. C.....223 S. Main St., Pratt, Kan., ALR
JENNINGS, CHAS. W. (1931)..Highland Bldg., Pittsburgh, Pa., Op.
JEROME, WM. Z. (1931)
436 Fort Washington Ave., New York, N. Y., ALR
JOBSON, G. B. (1903)
West Park and Liberty Sts., Franklin, Pa., OALR
*JOHNSON, A. E. (1919).....Watertown, S. D., OALR
JOHNSON, H. R. (1913)..Masonic Temple, Fairmont, W. Va., OALP

JOHNSON, OTIS H. ..... Morehead City, N. C., OALR
JOHNSON, THOMAS HOLLAND (1923)
30 W. 59th St., New York City, N. Y., Op.
JOHNSTON, W. H. (1912)
1421 Chapala St., Santa Barbara, Cal., OALR
JOHNSTON, WILSON (1917) ..... 611 Lovejoy St., Portland, Ore., OALR
JONES, C. C. (1924) ..... Doctors Bldg., Cincinnati, O., ALR
*JONES, CECIL CAIN (1928) ..... Equitable Bldg., Des Moines, Ia., OALR
JONES, EDLEY HICKS (1928)
First Nat'l Bank Bldg., Vicksburg, Miss., OALR
JONES, EDMUND LLOYD (1926)
Wheeling Clinic, Wheeling, W. Va., OALR
JONES, E. L. (1913), First Nat'l Bk. Bldg., Cumberland, Md., OALR
JONES, E. W. ..... 44 Atlanta St., Lynn, Mass., Op.A
JONES, F. W. (1919) ..... 101 Broadway, Alton, Ill., OALR
JONES, L. W. (1925) ..... 16 N. Goodman St., Rochester, N. Y., OALR
*JONES, M. F. (1930) ..... 121 E. 60th St., New York, N. Y., ALR
JONES, U. G. (1927)
Corner Market and Boone Sts., Johnson City, Tenn., ALR
JONES, VINCENT L. (1928)
3511 Washington Ave., St. Louis, Mo., Op.
*JONES, WM. S. (1927), 522 Sheridan Rd., Menominee, Mich., OALR
JORDAN, GEO. T. (1921), 30 N. Michigan Blvd., Chicago, Ill., OALR
JOSEPHSON, EMANUEL M. (1929)
993 Park Ave., New York, N. Y., ALR
JOY, HAROLD HENRY (1926)
504 State Tower Bldg., Syracuse, N. Y., Op.
JOYNT, A. J. (1924) ..... Black Bldg., Waterloo, Ia., OALR
JOYNT, M. J. (1930) ..... Royal Bldg., Le Mars, Ia., ALR
JUDD, H. B. (1917) ..... 40 E. 61st St., New York City, N. Y., ALR
*JUDD, J. H. (1930) ..... Medical Arts Bldg., Omaha, Neb., Op.
JUDGE, HARRY V. (1920) ..... 46 Willett St., Albany, N. Y., Op.

K

KAHN, M. E. (1925) ..... 104 S. Michigan Ave., Chicago, Ill., ALR
KARKEET, ROBERT BUDD (1928)
1203 Selling Bldg., Portland, Ore., OALR
KAUFFMAN, A. B. (1922), 1000 Chimes Bldg., Syracuse, N. Y., ALR
KAUFMAN, A. SPENCER (1917)
1923 Spruce St., Philadelphia, Pa., ALR
*KEARBY, D. O. (1917)
709 Hume-Mansur Bldg., Indianapolis, Ind., ALR
KEATING, T. F. (1927) ..... 1042 Maccabee Bldg., Detroit, Mich., OALR
KEEFE, WM. J. (1929), 110 E. Sidney Ave., Mt. Vernon, N. Y., ALR
KEELER, J. C. ..... Medical Arts Bldg., Philadelphia, Pa., ALR
KEIM, W. F. (1924) ..... 25 Roseville Ave., New York, N. Y., ALR
KEIPER, G. F., JR. (1930)
500 S. Lucas Ave., Los Angeles, Cal., ALR
*KEITH, W. E. ..... 1000 Professional Bldg., Kansas City, Mo., OALR

KELLER, S. A.....Sioux Falls Clinic, Sioux Falls, S. D., OALR
KELLEY, ISAAC D., JR. (1928)
3720 Washington Blvd., St. Louis, Mo., ALR
KELLY, JOSEPH DOMINIC (1928)
114 E. 54th St., New York City, N. Y., OALR
KEMLER, JOS. (1922).....1908 Eutaw Pl., Baltimore, Md., OALR
KENDALL, M. RAYMOND (1919)....Hanna Bldg., Cleveland, O., ALR
KENNEDY, ELDRED WESTON (1928)
707 Gas and Elect. Bldg., Rochester, N. Y., Op.
KENNON, W. G. (1924).....Doctors' Bldg., Nashville, Tenn., OALR
KETTELKAMP, FRED O. (1926)
301 Ferguson Bldg., Colorado Springs, Colo., ALR
*KEY, BEN WITT (1914)....100 W. 59th St., New York, N. Y., Op.
KEY, SAM N.....Norwood Bldg., Austin, Tex., OALR
KEYS, MORTON J. (1921)
100 Jones Blk., Victoria, B. C., Canada, OALR
KIEFER, HUGO A. (1915), Brockman Bldg., Los Angeles, Cal., OALR
*KIEHLE, F. A.....406 Taylor St., Portland, Ore., OALR
KILBOURNE, P. H (1924).....Fidelity Med. Bldg., Dayton, O., OALR
KILLEEN, JOHN JOSEPH (1928)
104 S. Michigan Ave., Chicago, Ill., ALR
*KINCAID, J. H.....421 W. Church Ave., Knoxville, Tenn., OALR
KINCANNON, LEROY TAYLOR (1928)
The Woodward Bldg., Birmingham, Ala., OALR
*KING, CLARENCE (1910), Union Central Bldg., Cincinnati, O., Op.
KING, CHAS. R. (1920).....Spitzer Bldg., Toledo, O., OALR
*KING, ED.....1002 Carew Tower, Cincinnati, O., ALR
KING, G. L., SR.....537 E. Market St., Alliance, O., OALR
KING, GEO. L., JR. (1929).....537 E. Market St., Alliance, O., Op.
KING, J. J. (1916).....30 E. 40th St., New York City, N. Y., ALR
KING, O. H. (1916)....Medical Arts Bldg., Hot Springs, Ark., OALR
KINNE, SANDFORD HALSEY (1928)
73 Main St., Binghamton, N. Y., ALR
*KIRBY, D. B. (1924).....30 W. 59th St., New York, N. Y., Op.
KIRKPATRICK, SAMUEL.....902 Alabama Ave., Selma, Ala., OALR
KISER, J. D.....Central Nat'l Bank Bldg., Bartlesville, Okla., OALR
KISTLER, O. F.....43 N. Franklin St., Wilkes-Barre, Pa., Op. AR
KISTNER, F. B.....600 Mayer Bldg., Portland, Ore., OALR
KITTELSON, THEODORE (1915)
Iverson and Lee Bldg., Fergus Falls, Minn., OALR
*KITTREDGE, FRANK E. (1931), Masonic Temple, Nashua, N.H., ALR
KLEINERT, MARGARET NOYES
82 Commonwealth Ave., Boston, Mass., ALR
KLINE, H. G. (1912).....State Tower Bldg., Syracuse, N. Y., ALR
KLINE, O. R. (1930).....414 Cooper St., Camden, N. J., ALR
KLINEDINST, JOHN FRED. (1906)
220 S. George St., York, Pa., Op.A
KLINGNER, THOMAS O. (1920)
318 Landers Bldg., Springfield, Mo., OALR
KNAPP, ARNOLD.....10 E. 54th St., New York, N. Y., Op.

*KNAPP, B. J. ..... Citizens' Bank Bldg., Evansville, Ind., OALR
KNAPP, F. N. (1925) ..... Bradley Bldg., Duluth, Minn., OALR
KNAUER, WM. J. (1930) ..... Buckman Bldg., Jacksonville, Fla., ALR
*KNEEDLER, G. C. (1912) ..... Professional Bldg., Pittsburgh, Pa., ALR
KNIGHTON, WILLIS S. (1931), 40 E. 61st St., New York, N. Y., Op.
KNODE, A. R. (1916) ..... 708 Medical Arts Bldg., Omaha, Neb., ALR
KNOWLTON, CHAS. D. (1929)
520 Commonwealth Ave., Boston, Mass., ALR
*KOEBBE, EZRA EDWIN (1929) ..... Columbus, Neb., ALR
KOENIG, IVAN J. (1928) ..... 40 North St., Buffalo, N. Y., Op.
*KOOBS, H. J. G. (1920) ..... 117½ W. Walnut St., Rogers, Ark., OALR
*KOPETZKY, S. J. ..... 51 W. 73rd St., New York City, N. Y., ALR
KRAFT, OSCAR H. ..... 1518 N. Dearborn Pkwy., Chicago, Ill., Op.
KRAUSS, FREDERICK ..... 1930 Chestnut St., Philadelphia, Pa., OALR
KREBS, ADOLPH (1914) ..... Jenkins Arcade, Pittsburgh, Pa., Op.
KRESS, GEO. H. ..... 245 Bradbury Bldg., Los Angeles, Cal., OALR
*KREUTZ, G. C. (1930) ..... Henry Ford Hospital, Detroit, Mich., ALR
KRIEGER, G. M. ..... 701 Washington St., Michigan City, Ind., OALR
KRIEGER, WM. A. (1919), 35 Market St., Poughkeepsie, N. Y., OALR
KRIMSKY, JOSEPH (1928)
263 Eastern Parkway, Brooklyn, N. Y., OALR
*KRONFELD, PETER C. (1931) ..... 950 E. 59th St., Chicago, Ill., Op.
KRUG, ERNEST F. (1908) ..... 988 Fifth Ave., New York, N. Y., Op.
KRUSKAL, I. D. (1925) ..... 863 Prospect Pl., Brooklyn, N. Y., Op.
*KUHN, H. S. (1930) ..... First Trust Bldg., Hammond, Ind., Op.
*KUHN, HUGH A. (1928) ..... First Trust Bldg., Hammond, Ind., ALR
KULLY, B. M. (1922) ..... Medical Arts Bldg., Omaha, Neb., ALR
KULVIN, MAX M. (1931) ..... 1 N. Crawford Ave., Chicago, Ill., ALR

L

LAESSLE, HENRY A. (1922)
The Lenox, 13th and Spruce Sts., Philadelphia, Pa., ALR
LAFF, H. I. (1930) ..... Metropolitan Bldg., Denver, Colo., ALR
LA FORCE, B. D. ..... 1140 Beverley Way, Altadena, Cal., OALR
LA FORCE, E. F. (1908)
Iowa State Bank Bldg., Burlington, Ia., OALR
LAMB, HARVEY D. (1920)
826 Metropolitan Bldg., St. Louis, Mo., Op.
LAMB, HARRY H. (1929)
American Bank Bldg., Davenport, Ia., ALR
LAMB, ROBERT SCOTT ..... Stoneleigh Ct., Washington, D. C., Op.
LAMPERT, MAX T. (1931), 55 E. Washington St., Chicago, Ill., ALR
*LANCASTER, W. B. (1913)
520 Commonwealth Ave., Boston, Mass., Op.
LANDFRIED, C. J. ..... 607 Baronne Bldg., New Orleans, La., ALR
LANDIS, J. E. ..... 517 Walnut St., Reading, Pa., ALR
LANING, J. H. (1918) ..... 907 Rialto Bldg., Kansas City, Mo., ALR
LAPSLEY, ROBERT M. (1902) ..... Masonic Temple, Keokuk, Ia., OALR
*LARGE, S. H. (1904) ..... Hanna Bldg., Cleveland, O., ALR

*LARKIN, BERNARD J. (1916)
Hume-Mansur Bldg., Indianapolis, Ind., Op.
LARSEN, CARL L. (1916)
1027 Lowry Medical Bldg., St. Paul, Minn., OALR
LARUE, C. L. (1922).....Highland Clinic, Shreveport, La., OALR
LASSALLE, ALBERT (1924), 3439 St. Denis St., Montreal, Can., OALR
LASSEN, FRITZ (1915).....Pueblo Clinic, Pueblo, Colo., OALR
LA VIGNE, A. A.....471 Park Ave., New York, N. Y., OALR
LAWRENCE, GRANVILLE A.
Medical Arts Bldg., Philadelphia, Pa., Op.
*LAWSON, LAWRENCE S. (1931), 636 Church St., Evanston, Ill., ALR
*LAWWILL, STEWART (1928)
Medical Arts Bldg., Chattanooga, Tenn., OALR
*LEASURE, JOHN K. (1931)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
LEAVENWORTH, R. O. (1930)
Lowry Medical Arts Bldg., St. Paul, Minn., Op.
*LEAVY, C. A.....Metropolitan Bldg., St. Louis, Mo., ALR
*LEBENSOHN, JAMES E. (1927)
25 E. Washington St., Chicago, Ill., OALR
LE COMPTE, E. D. (1927)
627 Boston Bldg., Salt Lake City, Utah, OALR
LEDERER, FRANCIS L. (1925)
30 N. Michigan Blvd., Chicago, Ill., ALR
LEECH, J. W.....369 Broad St., Providence, R. I., OALR
LEECH, V. M. (1930).....55 E. Washington St., Chicago, Ill., Op.
LEFLER, ANNA B. (1918), 610 S. Broadway, Los Angeles, Cal., OALR
LEHAN, JAMES W.....Park Place Bldg., 8th St., Greeley, Colo., OALR
*LEJEUNE, F. E. (1927)
632 Maison Blanche Bldg., New Orleans, La., ALR
LEMERE, H. B. (1912)
6381 Hollywood Blvd., Los Angeles, Cal., OALR
LEMOINE, A. N. (1920).....Rialto Bldg., Kansas City, Mo., Op.
LENKER, JOHN N. (1908).....1020 Huron Road, Cleveland, O., ALR
LENT, E. J.....122 N. Lafayette Blvd., South Bend, Ind., OALR
LERNER, MACY LEVI (1923), 332 Park Ave., Rochester, N. Y., Op.
LESHIN, NORMAN (1931)..180 N. Michigan Ave., Chicago, Ill., ALR
LESTER, HARRY S. (1909).....420 Murray Bldg., Streator, Ill., OALR
LEVENGOOD, H. WILSON (1927)
327 Wilshire Blvd., Santa Monica, Cal., OALR
LEVINE, JOSEPH (1931).....113 E. 61st St., New York, N. Y., Op.
LEVY, HARRY H.....1016 Fifth Ave., New York, N. Y., Op.
LEVY, LOUIS (1916).....1052 Madison Ave., Memphis, Tenn., ALR
LEVY, ROBERT (1896).....Metropolitan Bldg., Denver, Colo., ALR
LE WIN, THURBER (1929), 112 Linwood Ave., Buffalo, N. Y., Op.
LEWIS, A. C. (1916).....Exchange Bldg., Memphis, Tenn., OALR
LEWIS, EUGENE R. (1926), Roosevelt Bldg., Los Angeles, Cal., ALR
LEWIS, FIELDING O.....259 S. 17th St., Philadelphia, Pa., ALR
*LEWIS, F. PARK.....454 Franklin St., Buffalo, N. Y., Op.

LEWIS, GEORGE A. (1920).....Box 132, Roundup, Mont., OALR
LEWIS, J. D. (1921).....1534 State St., Santa Barbara, Cal., OALR
LEWIS, J. P. (1915).....543 Boylston St., Boston, Mass., ALR
LEWIS, PHILIP MERIWETHER (1928)
Exchange Bldg., Memphis, Tenn., OALR
LEWIS, WILLIAM W. (1921)
836 Lowry Bldg., St. Paul, Minn., OALR
*LEYDA, JAMES H. (1927), Metropolitan Bldg., Denver, Colo., ALR
*LICHTENBERG, J. S. (1906).....Rialto Bldg., Kansas City, Mo., Op.
LIDIKAY, C. J. (1920).....903 N. 7th St., Kansas City, Kan., OALR
*LIERLE, DEAN MCALLISTER (1928)
University Hospital, Iowa City, Ia., ALR
LIFSCHUTZ, JACOB (1925), 310 S. Michigan Ave., Chicago, Ill., OALR
LILLIE, HAROLD I.....Mayo Clinic, Rochester, Minn., ALR
LILLIE, W. IVAN (1930).....Mayo Clinic, Rochester, Minn., Op.
*LINGEMAN, B. N. (1930)
Ben Hur Bldg., Crawfordsville, Ind., ALR
*LINGEMAN, ED. L. (1929)
406 Penway Bldg., Indianapolis, Ind., ALR
LINN, J. G. (1919), 7026 Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*LISCHKOFF, M. A. (1913).....Blount Bldg., Pensacola, Fla., OALR
LITCHFIELD, J. T. (1921)
Medical Arts Bldg., Minneapolis, Minn., OALR
*LITTELL, JOSEPH J. (1931)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
LIVINGSTON, GEO. S. (1930), 185 N. Wabash Ave., Chicago, Ill., ALR
LLOYD, A. W. (1915).....804 Lloyd Bldg., Hammond, Ind., OALR
*LLOYD, RALPH I. (1925).....14 Eighth Ave., Brooklyn, N. Y., Op.
LOEB, CLARENCE (1907)
1054 Tower Rd., Hubbard Woods, Ill., Op.
LOFTUS, JOHN EDWARD (1921)
1930 Chestnut St., Philadelphia, Pa., ALR
LOGUE, LYLE J. (1931).....1304 Walker St., Houston, Texas, ALR
LOKEY, H. M.....Medical Arts Bldg., Atlanta, Ga., OALR
LOMBARDO, MELCHIORE (1923)
142 Joralemon St., Brooklyn, N. Y., Op.
LOOMIS, E. A. (1916)
Medical Arts Bldg., Minneapolis, Minn., OALR
LOOPER, EDWARD ANDERSON (1924)
104 Madison St., Baltimore, Md., OALR
LORIE, ALVIN J. (1920).....Commerce Bldg., Kansas City, Mo., ALR
LORING, J. BROWN.....25 E. Washington St., Chicago, Ill., OALR
LOVE, JOHN G. (1920).....Ilgenfritz Bldg., Sedalia, Mo., OALR
LOWELL, WM. H. (1931)
82 Commonwealth Ave., Boston, Mass., Op.
LOWRY, B. W. (1925).....205 Zack St., Tampa, Fla., OALR
LUBMAN, MAX (1919), 17 E. 96th St., New York City, N. Y., ALR
LUCAS, HAROLD ROY (1928)
Medical-Dental Bldg., Portland, Ore., OALR
LUEDDE, W. H. (1916).....Metropolitan Bldg., St. Louis, Mo., Op.

LUHR, ALFRED F. (1931)....40 West North St., Buffalo, N. Y., Op.
LUKENS, CHARLES (1906).....316 Michigan St., Toledo, O., OALR
LUKENS, J. A. (1930).....316 Michigan St., Toledo, O., ALR
LUKENS, ROBERT McD.....1923 Spruce St., Philadelphia, Pa., ALR
LUNDON, A. E. (1927)....1501 Crescent St., Montreal, Canada, ALR
LUONGO, ROMEO A. (1931)....1713 Pine St., Philadelphia, Pa., ALR
LUPTON, IRVING MARTIN (1928)
Medical Arts Bldg., Portland, Ore., OALR
LURIE, M. H. (1930).....483 Beacon St., Boston, Mass., ALR
LUX, PAUL (1925)....409 Commerce Bldg., Kansas City, Mo., ALR
*LYLE, DONALD J. (1927).....19 W. 7th St., Cincinnati, O., OALR
*LYMAN, HARRY W.(1923),906 Carleton Bldg., St. Louis, Mo., ALR
LYNCH, ROBT. J. (1931).....Equitable Bldg., Des Moines, Ia., ALR
LYONS, H. R.....30 N. Michigan Blvd., Chicago, Ill., ALR

Mc

MCALESTER, ANDREW W., JR., Rialto Bldg., Kansas City, Mo., Op.
MCALLISTER, J. C. (1910).....10 S. Mill St., Ridgway, Pa., AL
MCANDREWS, LEO F. (1931)
1930 Chestnut St., Philadelphia, Pa., Op.
MCBEAN, GEO. M.....58 E. Washington St., Chicago, Ill., OALR
MCBRIDE, W. O. (1917)
2902 S. Fairfield Ave., Ft. Wayne, Ind., OALR
*MCCANNEL, A. D.....33 Central Ave. W., Minot, N. D., OALR
MCCARTNEY, OLIVER P. (1920)
Valley Bank Bldg., Mesa, Ariz., OALR
MCCARTY, VIRGIL W.....Professional Bldg., Kansas City, Mo., OALR
*MCCASKEY, C. H.....Continental Bk. Bldg., Indianapolis, Ind., ALR
MCCAW, JOHN A.....418 Majestic Bldg., Denver, Colo., Op.
MCCLELLAN, W. E. (1927)
50 Farmington Ave., Hartford, Conn., OALR
MCCLELLAND, CARL C. (1923)
1857 David Whitney Bldg., Detroit, Mich., OALR
MCCONNELL, T. E., Logan Trust Bldg., New Kensington, Pa., OALR
MCCOOL, J. L. (1911).....450 Sutter St., San Francisco, Cal., Op.
MCCORMICK, T. F. (1930).....Wells Bldg., Milwaukee, Wis., ALR
MCCOY, J.....730 Fifth Ave., New York City, N. Y., ALR
MCCOY, L. L.....817 Summit Ave., Seattle, Wash., Op.
MCCREADY, J. HOMER.....121 University Pl., Pittsburgh, Pa., ALR
*MCCULLOCH, ROBERT J. P. (1920)
830 Medical Arts Bldg., Toronto, Can., OALR
MCCULLOUGH, C. J. (1929)
Washington Trust Bldg., Washington, Pa., ALR
MCDANNALD, C. E. (1927)
100 W. 59th St., New York, N. Y., Op.
MCDAVITT, BERTHA SCHENCK STECKER (1929)
Woodson Eye, Ear, Nose and Throat Hosp., Temple, Texas, ALR
*MCDERMOTT, JOHN J. (1924)
1821 Niles Ave., St. Joseph, Mich., OALR

McDIARMID, H. OLIVER (1918)
35 Clement Block, Brandon, Manitoba, Can., OALR
McDONALD, C. L.....964 Rose Bldg., Cleveland, O., OALR
McDONALD, DENNIS J. (1926)
17 E. 38th St., New York, N. Y., ALR
McDOUGALL, WM. L. (1931)
Atlanta Nat'l Bank Bldg., Atlanta, Ga., ALR
*McDOWELL, N. D. (1914)
275 Alexander St., Rochester, N. Y., OALR
McGARVEY, WM. ED. (1929)
Jackson City Bank Bldg., Jackson, Mich., ALR
McGILLICUDDY, O. B. (1930)
Metropolitan Bldg., Denver, Colo., ALR
McGINNIS, EDWIN.....104 S. Michigan Blvd., Chicago, Ill., ALR
McGIVERN, C. S. (1922), 109 S. Iowa Ave., Atlantic City, N. J., ALR
*McGREGOR, GREGOR (1931)..170 St. George St., Toronto, Can., ALR
McGUIRE, H. H.....105 N. Braddock St., Winchester, Va., OALR
McHENRY, D. D. (1912)
604 Medical Arts Bldg., Oklahoma City, Okla., OALR
McHENRY, L. C. (1930)
117 N. Broadway, Oklahoma City, Okla., ALR
McKEE, CLAUD W. (1917).....Coulter Bldg., Greensburg, Pa., OALR
*McKEE, GEO. J. (1922), Westinghouse Bldg., Pittsburgh, Pa., ALR
McKEE, JOSEPH WALLACE (1928)
Professional Bldg., Kansas City, Mo., Op.
McKEE, OTTO S.....83 Bryant St., Buffalo, N. Y., ALR
*McKEE, S. HANFORD (1910)
1528 Crescent St., Montreal, Can., Op.
McKELVEY, A. D.....160 Bloor St. W., Toronto, Can., ALR
McKENNAN, JAMES W.
514 Washington Trust Bldg., Washington, Pa., Op.
*McKEOWN, ELMER F.....Republic Bldg., Denver Colo., OALR
McKEOWN, HUGH S. (1931)..30 W. 59th St., New York, N.Y., Op.
*McKINNEY, A. R. (1922)
704 Second Nat'l Bank Bldg., Saginaw, Mich., Op.
McKINNEY, R.....899 Madison Ave., Memphis, Tenn., ALR
McLAUGHLIN, P. A., 71 S. Washington St., Wilkes-Barre, Pa., OALR
McLAURIN, JOHN G. (1926)
Medical Arts Bldg., Dallas, Tex., OALR
McLOONE, JOHN J. (1926)....611 Heard Bldg., Phoenix, Ariz., OALR
*McMAHON, BERNARD JOHN (1926)
Missouri Theater Bldg., St. Louis, Mo., ALR
*McMURRAY, J. B., Washington Trust Bldg., Washington, Pa., OALR
*McNALLY, WILLIAM JAMES
The Linton Apts., Montreal, Canada, ALR
McNICHOLS, W. A. (1925)
Dixon Nat'l Bank Bldg., Dixon, Ill., OALR
McRAE, JOHN H....Grand Rapids Clinic, Grand Rapids, Mich., Op.
McREYNOLDS, G. S.....Professional Bldg., Temple, Texas, ALR

McReynolds, John O.
907 Mercantile Bank Bldg., Dallas, Texas, OALR
*McWilliams, Chas. A. (1927)
G. and S. I. Bldg., Gulfport, Miss., ALR
MacCready, Paul B. (1931)
442 Temple St., New Haven, Conn., ALR
MacDonald, J. C. (1930)
301 W. 12th St., Oklahoma City, Okla., ALR
Macfarlane, P. B., Medical Arts Bldg., Hamilton, Ont., Can., OALR
Mackenzie, Alice V. (1919)
1724 Spruce St., Philadelphia, Pa., OALR
Mackenzie, Geo. W. (1912)
1724 Spruce St., Philadelphia, Pa., OALR
MacLachlan, A. A.....May Bldg., Pittsburgh, Pa., ALR
MacMillan, J. A. (1925), 1410 Stanley St., Montreal, Can., Op.
Macnie, J. S.....74 S. 11th St., Minneapolis, Minn., OALR

M

Madden, Reed.....Allen Bldg., Xenia, O., OALR
Maggard, D. I. (1920).....715 Orpheum Bldg., Wichita, Kan., OALR
Maghy, Chas. A. (1926)
Wilshire Medical Bldg., Los Angeles, Cal., Op.
Mahoney, Paul Leo (1928)
Donaghey Bldg., Little Rock, Ark., ALR
Maiden, S. D. (1918), 403 Bennett Bldg., Council Bluffs, Ia., OALR
Malone, F. B. (1930), Lubbock Sanitarium, Lubbock, Texas, ALR
*Mann, B. H. (1930)
N. E. corner 20th and Chestnut Sts., Philadelphia, Pa., Op.
*March, Clara A. (1922), 465 Ashland Ave., Buffalo, N. Y., Op.
Markel, J. Clyde.....Westinghouse Bldg., Pittsburgh, Pa., Op.
Marks, Julian B. (1929), 735 N. Water St., Milwaukee, Wis., ALR
Marks, Samuel Blackburn (1928)
Security Trust Bldg., Lexington, Ky., ALR
*Marlow, Searle B. (1924)
1004 State Tower Bldg., Syracuse, N. Y., Op.
Marquis, Geo. P.....30 N. Michigan Blvd., Chicago, Ill., ALR
Marsden, W. C. (1930), 64 N. Franklin St., Wilkes-Barre, Pa., ALR
Marshall, George Guerin.....Gryphon Bldg., Rutland, Vt., OALR
Marshall, Watson.....121 University Pl., Pittsburgh, Pa., ALR
Martin, Roy D. (1929).....Foote Bldg., Hastings, Neb., ALR
Martin, W. O., Jr. (1930), Medical Arts Bldg., Atlanta, Ga., Op.
*Mason, Roy Earl (1928).....Frisco Bldg., St. Louis, Mo., Op.
*Masters, Robert John (1928)
23 E. Ohio St., Indianapolis, Ind., Op.
Matheny, R. C.....306 E. Main St., Galesburg, Ill., OALR
Matheson, J. P. (1915).....6 West 7th St., Charlotte, N. C., ALR
Matthews, Justus..Medical Arts Bldg., Minneapolis, Minn., LR
Mauldin, L. O.
Chamber of Commerce Bldg., Greenville, S. C., OALR

MAUMENEE, A. E.....711 Empire Bldg., Birmingham, Ala., OALR
MAY, CHAS. H. (1904)
698 Madison Ave., New York City, N. Y., Op.
MAY, J. V. (1915).....1703 Main St., Marinette, Wis., OALR
MAYBAUM, J. L. (1916)
1079 Park Ave., New York City, N. Y., ALR
*MAYER, LEO LOEB (1928), 104 S. Michigan Ave., Chicago, Ill., Op.
MAYERBURG, EMIL R. (1929)
Medical Arts Bldg., Wilmington, Del., ALR
MEANOR, WM. C....619 Westinghouse Bldg., Pittsburgh, Pa., Op.
*MEANS, R. G. (1930).....137 East State St., Columbus, O., ALR
MEEK, RAYMOND E. (1931), 30 E. 40th St., New York, N. Y., Op.
MEIERHOF, E. L. (1912)
(Retired), The Westover, 253 W. 72nd St., New York, OALR
MELLINGER, WM. J. (1927)
120 E. Sola St., Santa Barbara, Cal., ALR
MELTON, BERNARD LEE, JR. (1931)
Goodrich Bldg., Phoenix, Ariz., Op.
MENGER, LOUIS C. (1929), 202 Weirfield St., Brooklyn, N. Y., ALR
MERCER, RAY (1920).....W. C. U. Bldg., Quincy, Ill., OALR
MERCER, R. E.....David Whitney Bldg., Detroit, Mich., LR
*MERICA, F. W. (1930).....14805 Detroit Ave., Lakewood, O., ALR
MERKEL, CHARLES CARSON (1928)
279 Rivard Blvd., Grosse Pointe Village, Mich., ALR
MERRILL, H. G. (1917)
35 East 2nd South St., South, Provo, Utah, OALR
MERTENS, PAUL STEARNS (1928)
1115 Bell Bldg., Montgomery, Ala., OALR
MESSIER, A. EUGENE (1925)...390 Main St., Worcester, Mass., OALR
MESSINGER, H. C.....210 Angell St., Providence, R. I., Op.
METZ, ROY B.....1214 Guardian Bldg., Cleveland, O., Op.
METZENBAUM, MYRON (1912)
Carnegie Medical Bldg., Cleveland, O., ALR
*MEYER, SAMUEL JAMES (1928)
58 E. Washington St., Chicago, Ill., Op.
MIDDLETON, A. B.....Sterry Block, Pontiac, Ill., OALR
MIDDLETON, HARRY E. (1920).....Times Bldg., Alton, Ill., OALR
MILLER, ALICE R. (1919)
418 Fern St., West Palm Beach, Fla., OALR
MILLER, C. E.....917 E. Main St., Muncie, Ind., OALR
MILLER, CLIFTON M...Stuart Circle Hospital, Richmond, Va., OALR
MILLER, H. ED. (1930).....Missouri Bldg., St. Louis, Mo., ALR
MILLER, HUGH.....Chambers Bldg., Kansas City, Mo., OALR
MILLER, JOHN.....121 East 60th St., New York, N. Y., ALR
MILLER, J. W. (1929).....305 E. 17th St., New York, N. Y., ALR
MILLER, THEODORE E. (1931)
122 S. Michigan Ave., Chicago, Ill., ALR
*MILLER, WM. H. (1931).....Tribune Bldg., Terre Haute, Ind., ALR
*MILLETTE, J. W.....Reibold Bldg., Dayton, O., OALR
MINER, S. G. (1896)....2641 E. Jefferson Ave., Detroit, Mich., ALR

*Minor, C. L.....First Nat'l Bank Bldg., Springfield, O., OALR
Minor, H. F.....Bank of Commerce Bldg., Memphis, Tenn., OALR
Minsky, Henry (1926)....1 West 80th St., New York, N. Y., Op.
Minton, Wm. H.....Carby Bldg., St. Joseph, Mo., OALR
Mitchell, E. K. (1915)
710 W. Lehigh Ave., Philadelphia, Pa., ALR
Mitchell, H. E. (1930)....15701 Detroit Ave., Cleveland, O., ALR
*Mithoefer, W.....Doctors Bldg., Cincinnati, O., ALR
Mittendorf, A. D.....339 Park Ave., New York, N. Y., Op.
Mittendorf, W. Keith.....115 E. 53rd St., New York, N. Y., Op.
Moise, Allyn B. (1918), Medical Arts Bldg., Shreveport, La., ALR
*Molt, Wm. F. (1911)
529 Bankers Trust Bldg., Indianapolis, Ind., ALR
Monaghan, D. G.....Majestic Bldg., Denver, Colo., Op.
*Monahan, J. A. (1913).....181 Chestnut St., Clinton, Mass., OALR
Monosmith, O. B. (1912).....O. P. S. Bldg., Lorain, O., OALR
Monson, S. H.....1322 B. F. Keith Bldg., Cleveland, O., OALR
Montgomery, D. Cameron (1928)
Leyser Bldg., Greenville, Miss., ALR
Moore, G. A. (1915).....353 Main St., Palmer, Mass., OALR
Moore, George H. (1919)
1737 Chestnut St., Philadelphia, Pa., OALR
Moore, John D. (1925)....6 Washington St., Bloomfield, N. J., OALR
*Moore, P. G. (1930).....Medical Arts Bldg., Cleveland, O., Op.
Moore, Paul McNary, Jr.
University Hospital, Iowa City, Ia., ALR
Moore, R. B. (1930).....Trinity Hospital, Little Rock, Ark., ALR
Moore, Roy Seeley (1928)
Medical Arts Bldg., Syracuse, N. Y., ALR
Moore, T. K.....Second Nat'l Bank Bldg., Akron, O., OALR
*Moore, T. W. (1901)....1050 Fifth Ave., Huntington, W. Va., OALR
Moore, William Frederick
255 S. 16th St., Philadelphia, Pa., ALR
Morgan, James A. (1916)
48 Young Bldg., Honolulu, T. Hawaii, OALR
Moriarty, Richard W. (1928)
Medical Center, White Plains, N. Y., OALR
Morley, G. A. (1930)
Crookston State Bank Bldg., Crookston, Minn., Op.
Morris, A. G. (1918).....277 Alexander St., Rochester, N. Y., ALR
Morrison, Wm. Wallace (1926)
39 E. 50th St., New York, N. Y., ALR
Morse, A. W.....507 Phoenix Bldg., Butte, Mont., OALR
Morsman, L. W. (1920)....Morsman Clinic, Hibbing, Minn., Op.
Morton, Howard McI.
1111 Nicollet Ave., Minneapolis, Minn., Op.
Morton, J. B. (1924).....90 E. Warren Ave., Detroit, Mich., OALR
*Mosher, Harris P.....828 Beacon St., Boston, Mass., ALR
*Moss, Harvey E.....Luhrs Bldg., Phoenix, Ariz., OALR

Moss, Olin Winn (1931)
611 Weber Bldg., Lake Charles, La., ALR
Mott, John S. 214 Argyle Bldg., Kansas City, Mo., OALR
Motto, M. P. (1929) Rose Bldg., Cleveland, O., Op.
Moulton, Everett C.
Merchants Nat'l Bank Bldg., Ft. Smith, Ark., OALR
Moulton, H. (1900)
Merchants Nat'l Bank Bldg., Ft. Smith, Ark., OALR
Moyle, Eugene H. (1931), 1088 Park Ave., New York, N.Y., ALR
*Mueller, Lillian B. (1931)
Continental Bank Bldg., Indianapolis, Ind., ALR
*Mullin, Wm. V. (1913)
Cleveland Clinic, Euclid Ave. at 93rd St., Cleveland, O., ALR
Mulsow, J. E. (1921)
231 West Wisconsin Ave. W., Milwaukee, Wis., OALR
*Muncaster, S. B. 1026 16th St., N. W., Washington, D. C., Op.
Muncy, Wm. M. (1914) 162 Angell St., Providence, R. I., OALR
*Mundt, G. Henry (1912)
25 E. Washington St., Chicago, Ill., OALR
Munson, F. T. (1930) 2201 E. Jefferson Ave., Detroit, Mich., ALR
Murphy, A. B. (1930) 2830 Grand Ave., Everett, Wash., ALR
Murphy, J. G. North Carolina Bank Bldg., Wilmington, N.C., OALR
Murray, A. N. 4753 Broadway, Chicago, Ill., OALR
Murray, Harry J. (1931) 53 South St., Stamford, Conn., ALR
Mussun, W. G. (1924) Medical Arts Bldg., Cleveland, O., ALR
Myers, Elmer A., 407 Board of Trade Bldg., Superior, Wis., OALR
Myers, Eugene Lee (1926), 207 Wall Bldg., St. Louis, Mo., ALR
*Myers, John L. (1920), 815 Shukert Bldg., Kansas City, Mo., OALR
Myerson, M. C. (1923) 12 E. 86th St., New York, N. Y., ALR

N

Nadeau, E. G. Bellin Bldg., Green Bay, Wis., OALR
*Naftzger, J. B. Davidson Bldg., Sioux City, Ia., OALR
Nance, Willis O. 30 N. Michigan Blvd., Chicago, Ill., OALR
Nash, C. Stewart (1929)
26 S. Goodman St., Rochester, N. Y., ALR
Neeper, Edw. R.
Ex. Nat'l Bank Bldg., Colorado Springs, Colo., Op.
Neher, Edwin Manson (1926)
Boston Bldg., Salt Lake City, Utah, Op.
Neighbors, Clarence A.
120 S. Los Angeles St., Anaheim, Cal., OALR
Nelson, Chas. F. 606 Schofield Bldg., Cleveland, O., OALR
Nelson, Leo A. (1930) 4105 Live Oak St., Dallas, Texas, ALR
Nelson, Allan Louis
830 Lowry Medical Arts Bldg., St. Paul, Minn., Op.
Nelson, Lloyd L. (1923) Fleming Bldg., Norfolk, Neb., OALR
Nemours, Paul R. (1929) 3852 Greer Ave., St. Louis, Mo., ALR

NEUMANN, WILLIAM HENRY (1928)
1005 N. 8th St., Sheboygan, Wis., OALR
*NEW, GORDON B.....Mayo Clinic, Rochester, Minn., L
NEWCOMB, JOHN R.
410 Hume-Mansur Bldg., Indianapolis, Ind., Op.
NEWELL, WM. S.....1029 Vermont Ave., Washington, D. C., Op.
*NEWHART, HORACE (1905)
Medical Arts Bldg., Minneapolis, Minn., ALR
*NEWTON, F. H. (1925)....907 Mercantile Bldg., Dallas, Texas, OALR
NEWTON, WM. B. (1931)..201 N. Second Ave., Alpena, Mich., OALR
NISSELSON, MAX.....754 Beck St., New York, N. Y., ALR
NOONAN, W. J. (1905).....4753 Broadway, Chicago, Ill., OALR
*NORCROSS, E. P. (1924).....8 S. Michigan Ave., Chicago, Ill., ALR
NORTON, ARTHUR H. (1931)..1162 Willamette, Eugene, Ore., Op.
NORTHCOTT, THOMAS A. (1926)
17 E. 38th St., New York, N. Y., Op.
*NOVAK, FRANK J. JR.....30 N. Michigan Blvd., Chicago, Ill., ALR
*NUGENT, OSCAR B. (1930)
231 W. Washington St., Chicago, Ill., Op.
*NUTTER, C. F.....16 Amherst St., Nashua, N. H., Op.

O

OAST, SAMUEL P., JR. (1929)
30 E. 40th St., New York, N. Y., Op.
OBERG, C. M.....Syndicate Bldg., Minneapolis, Minn., OALR
*O'BRIEN, C. S. (1924).....University Hospital, Iowa City, Ia., Op.
O'BRIEN, G. R. (1930)....307 Sterling Place, Brooklyn, N. Y., ALR
O'BRIEN, STEVE A. (1916)
1st Nat'l Bank Bldg., Mason City, Ia., OALR
O'CONNOR, D. F. (1918).....671 Broad St., Newark, N. J., OALR
O'CONNOR, RODERIC P.....1909 Franklin St., Oakland, Cal., Op.
ODENEAL, THOMAS H. (1929)....234 Cabot St., Beverly, Mass., OALR
OERTEL, T. E.....S. F. Bldg., Augusta, Ga., OALR
OESTERLING, H. E. (1922)
642 N. Interlachen Ave., Winter Park, Fla., OALR
OFFUTT, W. N. (1905)
406 Security Trust Bldg., Lexington, Ky., OALR
*O'HORA, BERNARD A. (1931)
8-266 General Motors Bldg., Detroit, Mich., ALR
*OLMSTEAD, WM. DEF. (1930)
1920 Pacific Ave., Atlantic City, N. J., Op.
OLSHO, S. L. (1912).....235 S. 15th St., Philadelphia, Pa., OALR
OPPEGAARD, C. L. (1930)..220 S. Broadway, Crookston, Minn., ALR
ORCUTT, D. C. (1912).....30 N. Michigan Ave., Chicago, Ill., Op.
O'REILLY, CHARLES A. (1911)
521 Medical Arts Bldg., Philadelphia, Pa., ALR
O'REILLY, W. F. (1913).....44 S. Common St., Lynn, Mass., OALR
O'ROURKE, D. H. (1927)....217 Imperial Bldg., Denver, Colo., Op.

ORR, ADRIAN V. B. (1929).....525 Welsh St., Chester, Pa., ALR
ORTON, HENRY B.....24 Commerce St., Newark, N. J., ALR
O'SULLIVAN, T. J. (1927).....10 Pine St., Portland, Me., ALR
OVERMAN, F. V.....Hume-Mansur Bldg., Indianapolis, Ind., ALR

P

PACE, ERRETT (1930).....City Nat'l Bank Bldg., Paducah, Ky., ALR
PACKARD, LOUIS A. (1926)
Haberfelde Bldg., Bakersfield, Cal., OALR
PAINTER, A. M. (1918)..Professional Bldg., Kansas City, Mo., ALR
PALMER, ARTHUR (1926)
667 Madison Ave., New York, N. Y., ALR
PALMER, BASCOM HEADEN (1928)
502 Huntington Bldg., Miami, Fla., OALR
PALMER, EDW. M. (1919)...629 Beacon Bldg., Wichita, Kan., OALR
PALMER, F. E. (1928).....301 Main St., Sterling, Colo., OALR
*PARKER, HARRY L. (1931).....88 East Ave., Lockport, N. Y., ALR
*PARKER, WALTER R.....David Whitney Bldg., Detroit, Mich., Op.
PARRAMORE, JAMES B. (1929)
St. James Bldg., Jacksonville, Fla., ALR
PARRISH, ROBERT E.....Medical Arts Bldg., San Antonio, Tex., ALR
PARSONS, J. G.....Crookston Clinic, Crookston, Minn., OALR
PATTERSON, ELLEN J. (1917)
121 University Pl., Pittsburgh, Pa., ALR
PATTERSON, W. E.....74 S. 11th St., Minneapolis, Minn., OALR
PAUL, WM. C. (1930)
Rhodes Medical Arts Bldg., Tacoma, Wash., ALR
PAYNE, A. L. (1908)
Union Nat'l Bank Bldg., Eau Claire, Wis., OALR
PAYNE, R. J. (1925).....University Club Bldg., St. Louis, Mo., ALR
PAYNE, V. LEROY (1930)
1018 Madison Ave., Memphis, Tenn., ALR
*PEARLMAN, S. J. (1925)....180 N. Michigan Ave., Chicago, Ill., ALR
*PEARSON, WM. W. (1908)
417 Bankers Trust Bldg., Des Moines, Ia., OALR
PEELER, C. N. (1920).....6 W. 7th St., Charlotte, N. C., OALR
PEER, LYNDON A.....965 Broad St., Newark, N. J., ALR
PEERY, VANCE PRICE (1929).....Kinston, N. C., ALR
PEIRCE, HOWARD W. (1923)
David Whitney Bldg., Detroit, Mich., ALR
*PELZ, MORT D. (1929).....Missouri Bldg., St. Louis, Mo., ALR
PEMBER, AUBREY H. (1926)
500 W. Milwaukee St., Janesville, Wis., OALR
PENDEXTER, RALPH S. (1926)
1424 K St., N. W., Washington, D. C., Op.
PENTECOST, R. S. (1926).....184 Bloor St., Toronto, Canada, OALR
PERSKY, A. H. (1930).....1922 Spruce St., Philadelphia, Pa., ALR
*PETER, L. C. (1914).....1930 Chestnut St., Philadelphia, Pa., Op.

PETERMAN, H. E. (1912)....518 Cathedral St., Baltimore, Md., OALR
PETERSON, R. A (1920).....150 Fifth Ave., New York, N. Y., OALR
*PETHERAM, CHAS. C. (1931)
Medical Arts Bldg., Portland, Ore., ALR
PFAFFLIN, C. A., 445 Bankers Trust Bldg., Indianapolis, Ind., OALR
PFINGST, A. O.....Heyburn Bldg., Louisville, Ky., Op.
PFINGSTEN, C. F. (1916)
6370 Waterman Ave., St. Louis, Mo., ALR
PHELPS, KENNETH A. (1920)
Medical Arts Bldg., Minneapolis, Minn., OALR
PHILLIPS, WM. LINTON.....131 Linwood Ave., Buffalo, N. Y., Op.
PHINNEY, F. D.....22 W. 7th St., Cincinnati, O., OALR
PINKERTON, F. J. (1929)
46 Young Hotel Bldg., Honolulu, Hawaii, OALR
PIPER, ARTHUR SYLVESTER (1928), 610 S. Monroe, Enid, Okla., OALR
PITKIN, C. E.....Carnegie Bldg., Cleveland, O., ALR
*PLUMER, JOHN SHAFFER (1928)
121 University Pl., Pittsburgh, Pa., Op.
POIRIER, GEO. H. (1931).....60 Bay State Rd., Boston, Mass., ALR
*POLLOCK, H. L (1911).....2551 N. Clark St., Chicago, Ill., ALR
PONTIUS, P. J.....1831 Chestnut St., Philadelphia, Pa., Op.
POOS, E. E. (1927).....Fisher Bldg., Detroit, Mich., OALR
POOS, GROVER HEWITT (1928)
University Club Bldg., St. Louis, Mo., OALR
POPE, E. C. (1930).....Woodward Bldg., Birmingham, Ala., ALR
PORTER, C. T. (1930)..520 Commonwealth Ave., Boston, Mass., ALR
PORTER, E. H. (1912).....85 Madison St., Tiffin, O., OALR
PORTER, L. B. (1913).....199 Thayer St., Providence, R. I., OALR
*POST, LAWRENCE T. (1920)
Metropolitan Bldg., St. Louis, Mo., Op.
POST, M. HAYWARD (1918)
Metropolitan Bldg., St. Louis, Mo., Op.
POST, WINFRED B. (1921)
First National Bank Bldg., Carthage, Mo., OALR
POSTLE, C. D. (1930).....240 E. State St., Columbus, O., ALR
POTTER, ALLEN B. (1929), 3725 Kingshighway, St. Louis, Mo., ALR
POTTER, GEO. B. (1921).....Medical Arts Bldg., Omaha, Neb., OALR
POTTER, L. F.....68 Walnut St., New Bedford, Mass., OALR
POTTER, PETER (1918).....Murray Hospital, Butte, Mont., OALR
POTTER, W. A. (1930)
7450 Grand River Ave., Detroit, Mich., ALR
*POTTER, W. W. (1927).....Medical Bldg., Knoxville, Tenn., OALR
POTTS, J. B.....Medical Arts Bldg., Omaha, Neb., ALR
POWELL, LYLE STEPHENSON (1928)
813 Mass. St., Lawrence, Kan., OALR
POWERS, EVERETT (1916)
Central Nat'l Bank Bldg., Carthage, Mo., OALR
PRANGEN, AVERY DE H. (1919), Mayo Clinic, Rochester, Minn., Op.
*PRATT, FRED J. (1912)
1753 Medical Arts Bldg., Minneapolis, Minn., OALR

*Pratt, John A. (1910)
1753 Medical Arts Bldg., Minneapolis, Minn., OALR
Prendergast, D. A.....Medical Arts Bldg., Cleveland, O., OALR
Preston, H. G. (1930), Professional Bldg., Harrisonburg, Va., ALR
Price, John B. (1929).....824 DeKalb St., Norristown, Pa., ALR
Price, Norman W. (1908), 445 3rd St., Niagara Falls, N. Y., OALR
Prince, Alexander L. (1928), 36 Pearl St., Hartford, Conn., Op.
*Proetz, A. W. (1923).....Beaumont Bldg., St. Louis, Mo., ALR
Proud, Willard C. (1920)
Phys. and Surg. Bldg., St. Joseph, Mo., OALR
Prout, A. W. (1920).....380 E. Town St., Columbus, O., OALR
Putnam, F. I.....501 St. Claire Bldg., San Jose, Cal., OALR
Pyle, Wallace (1915)....15 Exchange Pl., Jersey City, N. J., OALR

Q

Qualls, Harley Winfield (1928)
Exchange Bldg., Memphis, Tenn., OALR
Quinby, Wm. O'G. (1919).....14 James St., Newark, N. J., OALR
Quincy, J. E. (1927), 270 Commonwealth Ave., Boston, Mass., ALR
Quittner, S. S.....10515 Carnegie Ave., Cleveland, O., OALR

R

Radcliffe, McCluney, 1930 Chestnut St., Philadelphia, Pa., Op.
Ralls, C. T. (1916).....1st Nat'l Bank Bldg., Winfield, Kan., OALR
Ralston, Wallace (1918)
1300 Walker Ave., Houston, Texas, Op.
Ramsey, G. S. (1927), 1496 Mountain St., Montreal, Canada, Op.
Randall, Hiram (1928).....81 Main St., Binghamton, N. Y., ALR
*Ravdin, B. B.....Central Union Bk. Bldg., Evansville, Ind., OALR
Ravdin, M.....Central Union Bk. Bldg., Evansville, Ind., OALR
Ray, Victor (1901).....19 W. 7th St., Cincinnati, O., Op.
*Reaves, Charles R.
Reaves Infir., 117 W. Sycamore St., Greensboro, N. C., OALR
*Reaves, Robert Grisham (1928)
422 W. Cumberland Ave., Knoxville, Tenn., OALR
*Reaves, W. P. (1913)
Reaves Infir., 117 W. Sycamore St., Greensboro, N. C., OALR
Rector, A. E. (1915), 112 W. College Ave., Appleton, Wis., OALR
*Reed, Wm. W. (1930).....Mills Bldg., Topeka, Kan., ALR
Reeder, J. E. (1916).....Davidson Bldg., Sioux City, Ia., OALR
*Reese, Algernon B. (1928)
73 E. 71st St., New York City, N. Y., Op.
Reese, Monroe D. (1926).....123 N. 8th St., Lebanon, Pa., OALR
Reese, Warren S. (1921)....230 S. 21st St., Philadelphia, Pa., Op.
Reger, H. S. (1908).....Physicians Bldg., Jamestown, N. Y., OALR
Reiche, O. C. (1916).....Markle Bank Bldg., Hazleton, Pa., OALR
Reid, H. W. (1930).....19 Garfield Place, Cincinnati, O., Op.

REIM, HUGO.....417 Metropolitan Bldg., St. Louis, Mo., Op.
*REINEKE, GEO. F. (1926).....Box 137, New Ulm, Minn., OALR
RENAUD, G. L.....510 Joseph Campan Ave., Detroit, Mich., OALR
RENNER, W. S. (1900).....341 Linwood Ave., Buffalo, N. Y., ALR
*REPASS, R. E. (1920).....337 Lincoln Rd., Miami Beach, Fla., ALR
REULING, F. H. (1924).....Black Bldg., Waterloo, Ia., OALR
*REYNOLDS, H. G. (1909), City Nat'l Bk. Bldg., Paducah, Ky., OALR
REYNOLDS, J. S. (1918)
Medical Arts Bldg., Minneapolis, Minn., OALR
RHEIN, ALFRED ED. (1931)..Tribune Bldg., Terre Haute, Ind., ALR
*RHODE, SOLON LAFAYETTE (1928)
238 N. 6th St., Reading, Pa., OALR
RICE, CLARENCE PRENTICE.....Breckenridge, Minn., OALR
RICE, G. B. (1916).....99 Bay State Road, Boston, Mass., ALR
RICE, JOHN E. (1924).....390 Main St., Worcester, Mass., OALR
RICE, R. H. (1915).....331 Grove St., Milwaukee, Wis., OALR
*RICHARDS, L. G. (1924)..319 Longwood Ave., Boston, Mass., ALR
RICHARDSON, J. J. (1929)
Cook Hosp. and Beall Clinic, Fort Worth, Texas, ALR
RICHARDSON, S. A. (1925)
111 W. Adams St., Jacksonville, Fla., Op.
RICHIE, L. T. (1916).....McCormick Bldg., Trinidad, Colo., OALR
*RIDPATH, ROBERT F.
N. E. cor. 18th and Chestnut Sts., Philadelphia, Pa., LR
RIEGER, W. H. (1924).....Hanna Bldg., Cleveland, O., ALR
*RIGG, J. P. (1930)....Grand Island Clinic, Grand Island, Neb., ALR
RINDLAUB, ELIZABETH P.....de Lendrecie Bldg., Fargo, N. D., OALR
RINDLAUB, JOHN H.....595 E. Colorado St., Pasadena, Cal., OALR
RINGLE, CHAS. A. (1906).....Coronado Block, Greeley, Colo., ALR
RINGUEBERG, E. N. S. (1905), 13 Main St., Lockport, N. Y., Op.
*RISDON, E. F. (1924)....Medical Arts Bldg., Toronto, Canada, ALR
RISSER, EDWARD D. (1928), 310 Choate Bldg., Winona, Minn., OALR
ROBB, MAYO (1929).....323 Ellis Bldg., Phoenix, Ariz., ALR
*ROBERTS, S. E. (1916)..Professional Bldg., Kansas City, Mo., ALR
ROBERTS, WM. H.....65 N. Madison Ave., Pasadena, Cal., Op.
*ROBERTSON, EDW. N. (1918)
132½ W. 6th St., Concordia, Kan., OALR
ROBERTSON, F. D.....Ashton Bldg., Grand Rapids, Mich., OALR
ROBINSON, H. T. (1916).....Medical Bldg., Cumberland, Md., OALR
ROBINSON, R. E. (1920), 205½ E. Bremer Ave., Waverly, Ia., OALR
ROBISON, J. T. (1930).....309 State St., New London, Conn., ALR
ROGERS, A. S.....105 S. Jefferson Ave., Saginaw, Mich., OALR
ROGERS, J. T.....Medical Arts Bldg., Montreal, Can., ALR
ROGERS, ROBERT MERRIAM (1928)
680 St. John's Pl., Brooklyn, N. Y., Op.
ROLLER, LOUIS A. (1920)
446 Paris Ave., S. E., Grand Rapids, Mich., Retired
ROMMEL, JOHN C.....3701 N. Broad St., Philadelphia, Pa., Op.
ROMONEK, PHILIP L. (1929)..Med. Arts Bldg., Omaha, Neb., OALR

ROOKER, A. M. (1920).....Gluck Bldg., Niagara Falls, N. Y., ALR
ROSEBROUGH, F. H. (1929)....Moore Bldg., San Antonio, Tex., Op.
ROSENBAUM, J.....1396 St. Catherine's St. W., Montreal, Can., Op.
ROSENBAUM, MORRIS (1929), 604 E. 5th St., New York, N. Y., Op.
ROSENBERGER, H. C. (1929).....Rose Bldg., Cleveland, O., ALR
ROSENBLUTH, MICHAEL (1931)
1018 E. 163rd St., New York, N. Y., ALR
ROTH, ALBERT W. (1920), Medical Arts Bldg., Tulsa, Okla., OALR
ROTHSCHILD, HAROLD J....239 Lowry Bldg., St. Paul, Minn., OALR
ROTT, O. M. (1915)
Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
*ROW, GEORGE S. (1911)
906 Hume-Mansur Bldg., Indianapolis, Ind., Op.
ROWLAND, JOHN F. (1911)
Medical Arts Bldg., Hot Springs, Ark., OALR
ROWLAND, W. D. (1923)....220 Clarendon St., Boston, Mass., Op.
RUBENDALL, C. (1915).....Medical Arts Bldg., Omaha, Neb., OALR
RUBENSTEIN, A. H. (1930)...Med. Arts Bldg., Syracuse, N. Y., ALR
RUBY, F. M.....Union City, Ind., OALR
RUCKER, C. W. (1929)...Med. Arts Bldg., Minneapolis, Minn., Op.
*RUCKER, E. W., JR. (1916)
926 Woodward Bldg., Birmingham, Ala., ALR
RUDOLPHY, J. B. (1929).....1924 Pine St., Philadelphia, Pa., Op.
*RUEDEMANN, A. D. (1929)
Cleveland Clinic, Euclid Ave. and 93rd St., Cleveland, O., Op.
*RUNK, R. L. (1929)
Wash. Sanit. and Hosp., Takoma Pk., Washington, D. C., ALR
RUSH, C. C.....907 U. S. Nat'l Bk. Bldg., Johnstown, Pa., OALR
RUSSELL, J. A. MERLE (1922).....813 Sassafras St., Erie, Pa., ALR
RUSSELL, ROLAND D. (1931)
58 E. Washington St., Chicago, Ill., ALR
*RUSSELL, R. E. (1924)....601 Pioneer Bk. Bldg., Waterloo, Ia., OALR
RUST, E. G.....Hanna Bldg., Cleveland, O., Op.
*RUTHERFORD, C. W. (1925)
University Hospital, Dept. Ophth., Iowa City, Iowa, Op.
RYAN, ED. R. (1930).....735 N. Water St., Milwaukee, Wis., Op.
RYAN, L. M. (1927).....Wall Bldg., St. Louis, Mo., ALR
RYCHENER, RALPH ORLANDO (1928)
1720 Exchange Bldg., Memphis, Tenn., Op.
RYDER, D. R.....151 Rock St., Fall River, Mass., ALR
*RYERSON, F. L. (1925).....Fine Arts Bldg., Detroit, Mich., OALR

S

SACCO, ANTHONY G. (1920)
440 New York Ave., Union City, N. J., ALR
SALINGER, SAMUEL (1920)
25 E. Washington St., Chicago, Ill., ALR
SALLANDER, F. W. (1919)
385 New Orpheum Bldg., Sioux City, Iowa, OALR

*SAMUELS, BERNARD (1929), 57 W. 57th St., New York, N. Y., Op.
SANDERS, JOHN GILLIS (1929)
Merchants Nat'l Bank Bldg., Mobile, Ala., ALR
SANDERSON, DAVID D. (1916), 914 Stuart Bldg., Lincoln, Neb., OALR
SANDERSON, H. H. (1916)
David Whitney Bldg., Detroit, Mich., OALR
SANOR, DANIEL GALBREATH, JR. (1928)
206 E. State St., Columbus, O., ALR
SARTAIN, PAUL J. (1921), 2006 Walnut St., Philadelphia, Pa., Op.
SAUER, W. E. (1904)....Beaumont Med. Bldg., St. Louis, Mo., ALR
*SAUER, W. W. (1910).....231 Third St., Marietta, O., OALR
*SAVERY, C. E.....206 Sherland Bldg., South Bend, Ind., OALR
SCAL, JOSEPH COLEMAN (1926)
44 W. 74th St., New York, N. Y., ALR
*SCALES, H. L. (1920).....Rorabaugh Bldg., Hutchinson, Kan., OALR
SCALES, J. L. (1915).....Medical Arts Bldg., Shreveport, La., OALR
SCARLETT, HUNTER W. (1919)
230 S. 21st St., Philadelphia, Pa., Op.
*SCHALL, LEROY A. (1927)
270 Commonwealth Ave., Boston, Mass., ALR
SCHAPPERT, N. L. (1916)
59 S. Washington St., Wilkes-Barre, Pa., OALR
SCHENCK, C. P. (1920), Med. Arts Bldg., Fort Worth, Tex., OALR
*SCHIEFELBEIN, HARRY T. (1931)
Stevens Clinic Hospital, Welch, W. Va., Op.
SCHILD, E. H. (1906).....Brant Bldg., Canton, O., Op.
SCHILLER, A. N. (1918)
101 Central Park, West, New York, N. Y., ALR
SCHLANSER, MAJOR A. E. (1921)
Walter Reed Gen. Hosp., Army Med.Center,Washington,D.C., OALR
SCHLEGEL, HERMAN T. (1916), 501 Third St., Wausau, Wis., OALR
SCHLINDWEIN, G. WM.....138 W. 9th St., Erie, Pa., OALR
SCHLIVEK, KAUFMAN (1925)
1016 Fifth Ave., New York, N. Y., Op.
SCHLUER, E. P. (1930).....Metropolitan Bldg., St. Louis, Mo., ALR
SCHNECK, NATHAN (1931)
815 S. Eighth St., Manitowoc, Wis., ALR
SCHNEIDER, ANTON S.(1928), 56 Court St., Plattsburg, N. Y., Op.A
SCHOLZ, R. P.....Metropolitan Bldg., St. Louis, Mo., ALR
SCHRANT, J. H. (1916).....14 W. 1st St., Hutchinson, Kan., OALR
SCHULZE, E. C. (1926).....Beasley Bldg., Beeville, Texas, OALR
SCHURTER, M. A. (1924)....114 E. 7th St., Long Beach, Cal., OALR
SCHUSTER, FRANK P. (1927)
2000 N. Mesa Ave., El Paso, Tex., OALR
SCHUSTER, S. A. (1925).....1st Nat'l Bk. Bldg., El Paso, Tex., ALR
SCHUTZ, W. H. (1921).....Bryant Bldg., Kansas City, Mo., OALR
SCHWARTZ, A. A. (1927)
941 Park Ave., New York City, N. Y., ALR

SCHWARTZ, FREDERICK O. (1919)
Metropolitan Bldg., St. Louis, Mo., Op.
SCHWARTZ, V. J. (1929), Med. Arts Bldg., Minneapolis, Minn., ALR
SCHWARTZ, WILLIAM A. (1928)
Goodrich Bldg., Phoenix, Ariz., OALR
SCHWEINITZ, GEO. E. DE (1905)
1705 Walnut St., Philadelphia, Pa., Op.
*SCIARRETTA, SYLVIO A. (1931)
55 East Washington St., Chicago, Ill., ALR
SCOTT, MERLE E. (1926).....1st Nat'l Bk. Bldg., Massillon, O., OALR
SCROGGS, J. J. (1921).....218 E. Third St., Beaver, Pa., Op.A
*SEARCY, H. B. (1912).....2501 6th St., Tuscaloosa, Ala., OALR
SEAY, D. E.....907 Mercantile Bk. Bldg., Dallas, Texas, OALR
SEBERT, L. J. (1930).....Medical Arts Bldg., Toronto, Canada, Op.
SECOY, F. L. (1922).....301 Davidson Bldg., Sioux City, Ia., OALR
SEDGWICK, O. W. (1917).....Ionia, Mich., OALR
SEDWICK, W. A.....Republic Bldg., Denver, Colo., Op.
SEEGMAN, SIMON (1918).....Medical Bldg., Pittsburgh, Pa., ALR
SEELYE, W. K. (1920).....1215 4th Ave., Seattle, Wash., OALR
SELIGSTEIN, MILTON B. (1926)
801 Union and Planters Bk. Bldg., Memphis, Tenn., OALR
SELLERS, L. M.....Medical Arts Bldg., Dallas, Texas, ALR
*SELTZ, SAMUEL (1931), Provident Bank Bldg., Cincinnati, O., ALR
SENER, WALTER J.....Physicians Bldg., Philadelphia, Pa., Op.
SENSENEY, EUGENE TOWNER (1928)
316 Lister Bldg., St. Louis, Mo., ALR
SETTEL, NATHAN (1931).....250 W. 94th St., New York, N. Y., ALR
SEWALL, E. C. (1910)
Stanford Univ. Hospital, San Francisco, Cal., ALR
SEWARD, JACKSON A. (1931)
121 E. 60th St., New York, N. Y., ALR
SEYDELL, E. M. (1916)
1030 First Nat'l Bank Bldg., Wichita, Kan., ALR
*SHACKLETON, W. E. (1918)
2323 Prospect Ave., Cleveland, O., Op.
SHAFER, J. J. (1913).....Starks Bldg., Louisville, Ky., OALR
SHAHAN, W. E.....Metropolitan Bldg., St. Louis, Mo., Op.
SHANKLIN, E. M.....575 Hohman St., Hammond, Ind., OALR
SHANNON, C. E. G.....1930 Chestnut St., Philadelphia, Pa., Op.
SHANNON, CLARENCE WM. (1931)
1215 4th Ave., Seattle, Wash., Op.
SHAPERO, I. M. (1919).....289 Alexander St., Rochester, N. Y., OALR
SHAPIRO, S. L. (1930).....185 N. Wabash Ave., Chicago, Ill., ALR
SHARP, BENJ. S. (1926).....339 Thayer St., Providence, R. I., OALR
SHARRETT, GEO. O. (1921).....119 Bedford, Cumberland, Md., OALR
SHASTID, T. H. (1913).....Sellwood Bldg., Duluth, Minn., Op.
SHAWHAN, GLENN E. (1913)
Smallwood Bldg., Nampa, Idaho, OALR
*SHEA, J. J. (1919).....1018 Madison Ave., Memphis, Tenn., ALR

SHEAHAN, WM. L. (1926), 59 College St., New Haven, Conn., OALR
SHELLMAN, J. L. (1917)
Lowry Medical Arts Bldg., St. Paul, Minn., OALR
SHEMELEY, WM. G., JR., 1724 Spruce St., Philadelphia, Pa., OALR
SHERMAN, MAJ. A. ED., M. C., U. S. A. (1913)
Hdqts. U. S. Troops, Air Corp., Municipal Air Port,
Long Beach, Cal., OALR
SHERMAN, ELBERT S. (1912)....671 Broad St., Newark, N. J., Op.
SHIELDS, J. M. (1915), 264 Metropolitan Bldg., Denver, Colo., Op.
SHIELDS, LAWRENCE.....Allen Bldg., Xenia, O., ALR
SHIRAS, HOWARD H. (1920)
624 Guardian Bldg., Cleveland, O., Op.
*SHIREY, O. M. (1925).....Osborn Bldg., Cleveland, O., ALR
SHOEMAKER, FERDINAND, U. S. Veterans' Hospital No. 67,
Excelsior Springs, Mo., OALR
SHOEMAKER, S. A.....30 E. Church St., Orlando, Fla., OALR
SHOEMAKER, W. A. (1896)
1006 Carleton Bldg., St. Louis, Mo., Op.
SHREVE, OWEN M. (1905).....162 W. 8th St., Erie, Pa., Op.
SHULTS, E. J. G. (1920).....Haines Bldg., Eldorado, Kan., OALR
SHUMAN, G. H. (1917).....Park Bldg., Pittsburgh, Pa., Op.
SHUMATE, D. L.....500 Argyle Bldg., Kansas City, Mo., OALR
*SHURLY, BURT R.....62 Adams Ave., W., Detroit, Mich., ALR
SHUSTER, B. H. (1929)....1930 Chestnut St., Philadelphia, Pa., ALR
SHUTE, D. K.....1727 DeSales St., N.W., Washington, D. C., Op.
SIDWELL, C. E. (1929).....608 4th Ave., Longmont, Colo., ALR
*SIEGEL, FRANCIS X. (1922)
901 Union Central Life Bldg., Cincinnati, O., OALR
SILBERNAGEL, C. E. (1918).....240 E. State St., Columbus, O., OALR
SIMON, F. C. (1929).....914 Arcade Bldg., St. Louis, Mo., ALR
SIMONDS, O. F.....Hanna Bldg., Cleveland, O., Op.
SIMONS, N. S. (1930).....MacCornack Clinic, Whitehall, Wis., ALR
*SIMPSON, GEO. V. (1931), 1710 R. I. Ave., Washington, D. C., Op.
SIMPSON, H. LEE (1921)
1409 David Whitney Bldg., Detroit, Mich., ALR
SIMPSON, JOHN REID.....825 Highland Bldg., Pittsburgh, Pa., ALR
*SIMPSON, M. B. (1930)..Professional Bldg., Kansas City, Mo., ALR
*SIMPSON, W. LIKELY (1911)
Phys. and Surg. Bldg., Memphis, Tenn., ALR
SINAMARK, ANDREW (1928), 205 Power Bldg., Hibbing, Minn., OALR
SINGLETON, E. M. (1897)
111 E. Church St., Marshalltown, Ia., R
SISSON, RAYMOND J. (1928)
753 David Whitney Bldg., Detroit, Mich., Op.
*SKILLERN, S. R. JR. (1925), 1734 Pine St., Philadelphia, Pa., ALR
SKIRBALL, JOSEPH J. (1927)
353 Commonwealth Ave., Boston, Mass., Op.
SLEIGHT, R. D. (1906).....303 Post Bldg., Battle Creek, Mich., Op.
SLOAN, HENRY L. (1919).....6 W. 7th St., Charlotte, N. C., Op.
SLOANE, H. O. (1930).....1717 Pine St., Philadelphia, Pa., Op.

*Slocum, George (1906)
First Nat'l Bank Bldg., Ann Arbor, Mich., Op.
Smit, Wm. Maurice (1928)
719 Missouri Theater Bldg., St. Louis, Mo., ALR
Smith, Arthur E.....Donaldson Bldg., Minneapolis, Minn., OALR
*Smith, Carlie Souter (1927)
214 N. Jefferson Ave., Springfield, Mo., OALR
Smith, Carroll L. (1929)
Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
Smith, Dorland (1910), 836 Myrtle Ave., Bridgeport, Conn., Op.A
Smith, D. S. (1917).....State Bank Bldg., La Crosse, Wis., OALR
Smith, E. Terry.....36 Pearl St., Hartford, Conn., Op.
Smith, George B. (1914).....Harbin Hospital, Rome, Ga., OALR
Smith, H. A. (1925)....315 E. Philadelphia St., Whittier, Cal., OALR
Smith, Jas. M.....123 East 53rd St., New York, N. Y., ALR
Smith, J. M.....134½ N. Patterson St., Valdosta, Ga., OALR
Smith, R. C.....Board of Trade Bldg., Superior, Wis., OALR
*Smith, R. N. (1929).....Medical Arts Bldg., Tulsa, Okla., ALR
Smith, Stanley.....121 University Pl., Pittsburgh, Pa., Op.
Smith, Victor C. (1912)
1707 Pere Marquette Bldg., New Orleans, La., Op.
*Smith, Wm. L. (1930)
Medical Arts Bldg., Salt Lake City, Utah, Op.
*Smyth, Duncan C. (1931)
375 Commonwealth Ave., Boston, Mass., ALR
Snapp, Carl Foster (1928)
604 Medical Arts Bldg., Grand Rapids, Mich., ALR
Sneed, Carl M.....506 W. Broadway, Columbia, Mo., OALR
Snell, Albert C. (1925)
53 S. Fitzhugh St., Rochester, N. Y., Op.
Snelling, P. W. (1931).....179 Allyn St., Hartford, Conn., ALR
Snyder, W. H.....2001 Callingwood Ave., Toledo, O., OALR
Somberg, J. S. (1923).....470 Park Ave., New York, N. Y., Op.
*Sonnenschein, R. (1915)
180 N. Michigan Ave., Chicago, Ill., ALR
*Spaeth, Edmund B. (1928)
1930 Chestnut St., Philadelphia, Pa., Op.
Spake, LaVerne B. (1920)
322 Brotherhood Bldg., Kansas City, Kan., ALR
Spalding, J. A. (1896).....627 Congress St., Portland, Me., Op.A
*Spencer, Frank R. (1904), Physicians Bldg., Boulder, Colo., OALR
Spencer, George Albert (1928)
1127 Eleventh St., Sacramento, Cal., OALR
Spencer, S. C. (1929).....Professional Bldg., Waco, Texas, Op.
Spencer, W. H.....1830 S. Rittenhouse Sq., Philadelphia, Pa., ALR
Spengler, John Arthur (1916)
423 S. Main St., Geneva, N. Y., Op.
Spiesman, I. G. (1929)....25 E. Washington St., Chicago, Ill., ALR
Spitze, E. C. (1916).....Murphy Bldg., East St. Louis, Ill., Op.

SPROULE, RALPH P. (1928)
Goldsmith Bldg., Milwaukee, Wis., OALR
ST. CLAIR, C. T. (1930)
Corner Bland and Ramsey Sts., Bluefield, W. Va., ALR
STAHLMAN, FRED C.....Commercial Bldg., Waynesburg, Pa., OALR
*STANBERRY, HENRY.....556 Doctors Bldg., Cincinnati, O., Op.
STANFORD, J. B.....1052 Madison Ave., Memphis, Tenn., OALR
STANLEY, C. R. (1930).....Worthington, Minn., ALR
*STANTON, HARMON LAMONTTE (1928)
Central Union Bldg., Evansville, Ind., OALR
STARK, JESSE B. (1925).....45 Park Ave., New York, N. Y., Op.
STARK, WM. B. (1927).....Mayo Clinic, Rochester, Minn., ALR
STAUFFER, FRED (1912)
Medical Arts Bldg., Salt Lake City, Utah, OALR
STEELE, WILLARD (1928)
Provident Bldg., Chattanooga, Tenn., OALR
STEIM, J. M. (1914)
First Nat'l Bank Bldg., New Kensington, Pa., Op.
STEINFELD, A. L. (1922).....125 15th St., Toledo, O., OALR
STEVENSON, D. W.....165 E. Market St., Akron, O., OALR
STEVENSON, WALTER (1915).....W. C. U. Bldg., Quincy, Ill., OALR
*STEVENSON, WM. W. (1931).....Kresge Bldg., Flint, Mich., ALR
STEWART, WALTER E. (1920)
Tribune Bldg., Terre Haute, Ind., OALR
STIEREN, E. (1905)....626 Union Trust Bldg., Pittsburgh, Pa., Op.
STILES, CHAS. M. (1908)....1130 Spruce St., Philadelphia, Pa., Op.
STIMSON, GEORGE W. (1919).....Packard Bldg., Warren, O., OALR
STINSON, WM. DAVID (1928)
805 Medical Arts Bldg., Memphis, Tenn., ALR
STITT, H. L. (1927).....656 Doctors Bldg., Cincinnati, O., OALR
STOCKLER, J. A. (1917).....332 North Fifth St., Reading, Pa., OALR
*STOKES, WM. H. (1926).....Medical Arts Bldg., Omaha, Neb., Op.
*STOLL, KARL L. (1905).....19 West 7th St., Cincinnati, O., ALR
*STONE, H. B. (1930).....Medical Arts Bldg., Roanoke, Va., ALR
STONECYPHER, D. D.....107 S. 9th St., Nebraska City, Neb., ALR
STOTTER, ARTHUR L. (1916), 1148 Euclid Ave., Cleveland, O., OALR
STOUT, PHILIP S.....269 S. 19th St., Philadelphia, Pa., ALR
STOUTENBOROUGH, WM. A. (1927)
21 East State St., Columbus, O., OALR
STRADER, GEO. L. (1904)....408 Hynds Bldg., Cheyenne, Wyo., OALR
STRAUSS, J. F. (1925).....104 S. Michigan Blvd., Chicago, Ill., ALR
*STREIT, AUGUST J. (1931).....Fisk Bldg., Amarillo, Texas, ALR
STRICKLER, D. A.....Republic Bldg., Denver, Colo., Op.
STRONG, J. C. (1915).....810 Harrison Ave., Leadville, Colo., OALR
STROUD, EDWARD FRANK (1926)
Medical Professional Bldg., Corpus Christi, Texas, ALR
STROUT, E. S. (1907).....74 S. 11th St., Minneapolis, Minn., OALR
STROUT, G. E. (1921), 1009 Nicollet Ave., Minneapolis, Minn., OALR
STUEBER, F. G. (1905).....209 W. North St., Lima, O., OALR
STUEBER, PAUL J. (1917).....209 W. North St., Lima, O., OALR

STURGES, LEIGH F. (1920), 121 E. 60th St., New York, N. Y., ALR
STURM, S. A. (1916)
7036 Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
*SUKER, GEO. F. (1896)....25 E. Washington St., Chicago, Ill., Op.
SULLIVAN, J. J., JR. (1923)
228 Scranton Life Bldg., Scranton, Pa., ALR
SULZER, G. A. (1907).....327 E. State St., Columbus, O., OALR
*SULZMAN, FRANK MALCOLM (1922)
1831 Fifth Ave., Troy, N. Y., OALR
*SUMMERS, WM. S. (1927)
1613 David Whitney Bldg., Detroit, Mich., OALR
SUTHERLAND, J. M. (1929)
8-257 General Motors Bldg., Detroit, Mich., ALR
SWAB, CHARLES MARION (1928)
Medical Arts Bldg., Omaha, Neb., Op.
SWAN, C. J. (1911).....636 Church St., Evanston, Ill., OALR
SWEET, ROBERT B. (1926)
Pacific S. W. Bk. Bldg., Long Beach, Cal., OALR
SWERDFEGER, E. B. (1927), 1008 Republic Bldg., Denver, Colo., ALR
SWETNAM, C. R. K. (1915), Masonic Temple, Prescott, Ariz., ALR

T

TANGEMAN, H. F. (1919)
606 Union Central Bldg., Cincinnati, O., ALR
TAQUINO, GEORGE J. (1920)
208 Audubon Bldg., New Orleans, La., ALR
TARUN, WILLIAM.....104 West Madison St., Baltimore, Md., OALR
TASSMAN, ISAAC C. (1929)
Medical Arts Bldg., Philadelphia, Pa., Op.
TAYLOR, CHARLES B. (1921).....118 E. 2nd St., Ottumwa, Ia., ALR
TAYLOR, EDGAR M. (1931)
Medical Arts Bldg., Portland, Ore., Op.
TAYLOR, LILLIAN E. (1905)
25 E. Washington St., Chicago, Ill., ALR
TAYLOR, M. C. (1915).....1204 May Bldg., Pittsburgh, Pa., Op.
TEALL, RAYMOND EDWIN (1928)
Professional Bldg., Kansas City, Mo., ALR
TEBBUTT, H. K. (1930).....240 State St., Albany, N. Y., ALR
*THEOBALD, GEORGIANA DVORAK (1929)
715 Lake St., Oak Park, Ill., Op.
*THEOBALD, JOHN J. (1929)
58 E. Washington St., Chicago, Ill., ALR
THEOBALD, W. H. (1930)..307 N. Michigan Ave., Chicago, Ill., ALR
*THIGPEN, C. A.....401 S. Court St., Montgomery, Ala., OALR
THOMAS, C. D. (1914), Central Nat'l Bank Bldg., Peoria, Ill., OALR
THOMAS, FRANCIS W. (1916), 327 E. State St., Columbus, O., OALR
THOMAS, H. G. (1926).....411 30th St., Oakland, Cal., OALR
*THOMAS, JAMES W. (1928).....Sharp Bldg., Lincoln, Neb., OALR
THOMASHEFSKY, MILTON (1930)
135 Eastern Parkway, Brooklyn, N. Y., ALR

THOMASON, H. E.....Rialto Bldg., Kansas City, Mo., ALR
THOMASSON, W. J. (1910).....942 York St., Newport, Ky., ALR
THOMPSON, E. H.....8 W. 9th St., Cincinnati, O., OALR
THOMPSON, G. H. (1909).....74 North St., Pittsfield, Mass., OALR
THOMPSON, HOWARD E. (1928), 1100 Main St., Dubuque, Ia., OALR
THOMPSON, PETER H. (1914)
308 Commonwealth Ave., Boston, Mass., Op.
THOMSON, J. J. (1907)
531 East Lincoln Ave., Mt. Vernon, N. Y., ALR
THOMSON, VON COLDITZ, G.
25 E. Washington St., Chicago, Ill., OALR
THORPE, HARVEY ED. (1931)
8075 Jenkins Arcade Bldg., Pittsburgh, Pa., Op.
TILDERQUIST, D. L. (1925), 425 Bradley Bldg., Duluth, Minn., OALR
TILLOTSON, R. S. (1930)....Woodland Clinic, Woodland, Cal., ALR
TIMBERMAN, ANDREW.....Beggs Bldg., Columbus, O., OALR
TINDOLPH, L. W.....101 N. Union St., Olean, N. Y., ALR
TINGLEY, LOUISA PAINE (1913)
9 Massachusetts Ave., Boston, Mass., Op.
Also 171 Westminster St., Providence, R. I.
TITSWORTH, GUY.....111 W. 4th St., Sedalia, Mo., OALR
TIVNEN, RICHARD J.....30 N. Michigan Ave., Chicago, Ill., OALR
*TOBEY, GEO. L., JR.....270 Commonwealth Ave., Boston, Mass., ALR
TODD, GROVER CLEVELAND (1922)
6133 Jenkins Arcade Bldg., Pittsburgh, Pa., ALR
TOMASSENE, R. A. (1922)
Wheeling Bank and Trust Bldg., Wheeling, W. Va., OALR
TOMB, E. H. (1930).....129 Concord St., Framingham, Mass., ALR
*TOMLIN, W. S. (1912)
Hume-Mansur Bldg., Indianapolis, Ind., ALR
TOMS, S. W. S. (1909).....120 S. Broadway, Nyack, N. Y., OALR
TOOKER, C. W. (1907).....Carleton Bldg., St. Louis, Mo., Op.
*TOOT, J. F. (1930).....First Nat'l Bank, Canton, O., Op.
TOWNSEND, JOHN FERRARS (1923)
85 Wentworth St., Charleston, S. C., OALR
*TRIBLE, G. B. (1920)
1801 Eye St., N. W., Washington, D. C., OALR
TRIMBLE, CLARENCE S. (1920)
405 Citizens Nat'l Bank Bldg., Emporia, Kan., OALR
TRIPP, IRA A. (1920).....436 Rose Bldg., Cleveland, O., OALR
TRIPP, LEROY R.....Security Bldg., Sioux City, Ia., OALR
*TUCKER, GABRIEL (1923)....326 S. 19th St., Philadelphia, Pa., B
TUCKERMAN, W. C. (1911)..733 Osborn Bldg., Cleveland, O., Op.
TUCKERMAN, W. H.....733 Osborn Bldg., Cleveland, O., OALR
TURNBULL, F. M. (1916)
806 Pacific Mutual Bldg., Los Angeles, Cal., ALR
*TURNER, H. H. (1914), 902 Professional Bldg., Pittsburgh, Pa., Op.
TURNER, OLIVER W. (1909), Masonic Temple, Augusta, Me., OALR
TUSAK, ERVIN A. (1931)..115 East 90th St., New York, N. Y., Op.

U

UHLER, S. MANN (1930).....104 N. 8th St., Allentown, Pa., ALR
UPHAM, HELEN F. (1907), 305 3rd Ave., Asbury Park, N. J., OALR
UREN, C. T. (1919), 537 City Nat'l Bank Bldg., Omaha, Neb., ALR
URNER, M. H.....2600 Union Central Bldg., Cincinnati, O., OALR

V

VAIL, DERRICK T., JR. (1931).....24 E. 8th St., Cincinnati, O., Op.
*VAIL, H. H. (1924).....24 E. 8th St., Cincinnati, O., ALR
VALONE, FRANK H. (1931).....258 Genesee St., Utica, N. Y., ALR
VAN DELLEN, A. L. (1930) .6857 S. Halsted St., Chicago, Ill., Op.
VAN HORN, A. F. (1930)....514 Central Ave., Plainfield, N. J., ALR
VAN KIRK, FRANK J. (1920)
Bellingham Nat'l Bank Bldg., Bellingham, Wash., OALR
*VAN OSDOL, HARRY A. (1931)
828 Chamber of Commerce, Indianapolis, Ind., ALR
VAN POOLE, G. McD. (1925)
Young Bldg., Honolulu, T. Hawaii, OALR
VAUGHAN, G. E.....711 3rd St., Louisville, Ky., OALR
VAUGHN, E. H.....Citizens Nat'l Bank Bldg., Tyler, Tex., OALR
VEASEY, C. A., SR. (1905)
Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
*VEASEY, C. A. JR. (1927)
Paulsen Medical and Dental Bldg., Spokane, Wash., OALR
VOIGT, C. B. (1912).....1702 Broadway, Mattoon, Ill., OALR
*VON DER HEYDT, ROBERT (1926)
25 E. Washington St., Chicago, Ill., Op.

W

WACHSBERGER, ALFRED (1931)
51 W. 73rd St., New York, N. Y., ALR
*WAGENER, HENRY P. (1926)....Mayo Clinic, Rochester, Minn., Op.
WAGERS, ARTHUR J. (1920)
1429 Spruce St., Philadelphia, Pa., ALR
WAGNER, CARL B. (1912)..30 N. Michigan Ave., Chicago, Ill., OALR
*WAGNER, W. A. (1927)
914 American Bank Bldg., New Orleans, La., ALR
WALKER, CHAS. C.(1920), 604 Maccabee Bldg., Detroit, Mich., Op.
WALKER, C. E. (1920).....517 Jacobson Bldg., Denver, Colo., Op.
*WALKER, GEO. H. (1927).....172 Main St., Winona, Minn., OALR
WALKER, HERBERT S.....30 N. Michigan Ave., Chicago, Ill., OALR
WALKER, WALDO WEBSTER (1928)
99 Bay State Road, Boston, Mass., ALR
WALSH, JOSEPH MARK (1922).....Rapid City, S. D., OALR
WARD, J. W. (1930)..Exchange Bank Bldg., Greenville, Tex., ALR
*WARNER, ROBT. J. (1929).....Doctors Bldg., Nashville, Tenn., Op.
WARREN, WILLIAM C., JR. (1928)
618 Atlanta Nat'l Bank Bldg., Atlanta, Ga., ALR

WARWICK, HAROLD LYNWOOD (1928)
913 Medical Arts Bldg., Fort Worth, Texas, ALR
WATKINS, EDWIN DIAL (1926)
1412 Exchange Bldg., Memphis, Tenn., OALR
WATKINS, H. R. (1930)....Griesheim Bldg., Bloomington, Ill., ALR
WATSON, H. DEWITT (1915)
151 Front St., Binghamton, N. Y., OALR
WATSON, JAMES A.....74 S. 11th St., Minneapolis, Minn., OALR
*WATSON, R. S. (1923)..120 N. Michigan St., Saginaw, Mich., OALR
*WAUGH, J. M.
Cleveland Clinic, Euclid Ave. at 93rd St., Cleveland, O., OALR
WEAVER, JOHN O. (1929)
608½ W. Sheridan Ave., Shenandoah, Ia., ALR
WEAVER, T. W. (1914).....201½ N. Main St., Wichita, Kan., Op.
*WEBER, HENRY CHARLES (1928)
U. S. Naval Hospital, Great Lakes, Ill., OALR
WEBSTER, F. R. (1930)
720 State Tower Bldg., Syracuse, N. Y., Op.
WEED, HARRY M. (1908)....196 Linwood Ave., Buffalo, N. Y., Op.
WEEKS, JOHN E....360 Edgecliff Road, Portland, Ore., Retired, Op.
*WEEKS, WEBB W. (1923)
20 E. 53rd St., New York City, N. Y., Op.
WEETH, C. R. (1930).....37 Remsen St., Brooklyn, N. Y., ALR
WEIDLEIN, IVAN F. (1930).....Osborn Bldg., Cleveland, O., ALR
*WEIH, E. P. (1920).....Wilson Bldg., Clinton, Ia., OALR
WEIL, A. I.....3503 Prytania St., New Orleans, La., ALR
WEINBERGER, NELSON S. (1928)
Robert Packer Hospital, Sayre, Pa., OALR
WEINSTEIN, M. A. (1927), 1309 Spruce St., Philadelphia, Pa., ALR
WEISSER, E. A.....806 May Bldg., Pittsburgh, Pa., Op.
WELCH, ROBERT S. G. (1921)
46 State Circle, Annapolis, Md., OALR
WELLS, DAVID W. (1908), Hotel Westminster, Boston, Mass., Op.
WELLS, GEORGE S.....8 E. Arrellaga, Santa Barbara, Cal., ALR
WELTY, C. F. (1926).....210 Post St., San Francisco, Cal., ALR
WENCKE, CARL G.....Battle Creek Sanit., Battle Creek, Mich., ALR
*WENDEL, J. S. (1922)
744 David Whitney Bldg., Detroit, Mich., ALR
WENNER, W. T. (1930)....St. Mary's Bldg., St. Cloud, Minn., Op.
WESSELS, ANDREW B. (1929)
Medico-Dental Bldg., San Diego, Cal., ALR
WESTLAKE, SAMUEL B., Beaumont Med. Bldg., St. Louis, Mo., ALR
WETHERBEE, CHARLES (1926)
Booth Bldg., Twin Falls, Idaho, OALR
WETZEL, JOHN O. (1930)
Capitol Saving and Loan Bldg., Lansing, Mich., Op.
WEXLER, DAVID (1930).....55 E. 86th St., New York, N. Y., Op.
WEYMANN, M. F. (1929)
2007 Wilshire Blvd., Los Angeles, Cal., Op.

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750 Main St., Hartford, Conn., ALR
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WHELAN, G. L. (1925).....2100 Walnut St., Philadelphia, Pa., ALR
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*WHITAKER, J. (1917)
Hume-Mansur Bldg., Indianapolis, Ind., OALR
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WHITE, E. HAMILTON (1912)
1610 Sherbrooke St., W., Montreal, Canada, ALR
WHITE, O. E. (1930).....28 N. Main St., Cortland, N. Y., ALR
WHITE, VIVIAN P. (1931)
Ezra Thompson Bldg., Salt Lake City, Utah, ALR
WHITE, WILLIAM BEVERLY (1928)
Gurley Bldg., Stamford, Conn., OALR
WHITNEY, E. L. (1930)..Henry Ford Hospital, Detroit, Mich., ALR
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Bank of Kalamazoo Bldg., Kalamazoo, Mich., OALR
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WINTER, JOHN A. (1923).....205 W. 2nd St., Duluth, Minn., OALR
*WISE, RALPH C. (1914).....News Bldg., Mansfield, O., OALR
WOLCOTT, CHAS. C. (1931).....291 Broadway, Yonkers, N. Y., ALR
WOLD, K. C. (1924)
1027 Lowry Med. Arts Bldg., St. Paul, Minn., OALR
WOLF, SAMUEL (1929).....55 E. Washington St., Chicago, Ill., Op.
*WOLFE, CLAUDE T. (1916).....Heyburn Bldg., Louisville, Ky., OALR

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Y

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HESS, WM. L.....OALR

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BOURBON, OLIVER P.....520 W. 7th St., OALR
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BOYCE, WM A.....Roosevelt Bldg., Op.
BROWN, J. M.....1136 W. 6th St., ALR
CRANE, WALTER RAYMOND.....756 S. Broadway, ALR
DAYTON, G. O.....1401 S. Hope St., OALR
DETLING, FRANK E.....500 S. Lucas St., OALR
DOW, JULIAN N.....727 W. Seventh St., Op.
FISHER, CARL.....1930 Wilshire Blvd., Op.
GOSNEY, CHARLES W.....Medical Arts Bldg., Op.
HOPKINS, W. E.....321 S. Lorraine Blvd., OALR
IRVINE, A. RAY.....1144 Roosevelt Bldg., Op.
KEIPER, GEORGE F., JR.....500 S. Lucas Ave., ALR
KIEFER, HUGO A.....Brockman Bldg., OALR
KRESS, GEORGE H.....245 Bradbury Bldg., OALR
LEFLER, ANNA B.....610 S. Broadway, OALR
LEMERE, H. B.....6381 Hollywood Blvd., OALR
LEWIS, EUGENE R.....Roosevelt Bldg., OALR
MAGHY, CHARLES A.....Wilshire Medical Bldg., Op.
TURNBULL, F. M.....806 Pacific Mut. Bldg., ALR
WEYMANN, MORIE FREDERICK.....2007 Wilshire Blvd., Op.

OAKLAND

BEAUDOUX, HENRY ALEXANDER.....230 Grand Ave., OALR
O'CONNOR, RODERIC P.....1904 Franklin St., Op.
THOMAS, H. G.....411 30th St., OALR

PALOS VERDES ESTATES

STEIN, O. J.....ALR

PASADENA

CADY, D. W.....65 N. Madison Ave., Op.
CHAPMAN, VERNON A.....1275 E. Green St., OALR
HEITGER, J. D.....First Trust Bldg., OALR
HENNINGER, L. L.....Professional Bldg., OALR
RINDLAUB, JOHN H.....595 E. Colorado St., OALR
ROBERTS, WM. H.....65 N. Madison Ave., Op.

SACRAMENTO

BRIGGS, W. E.....1027 10th St., OALR
SPENCER, GEORGE ALBERT.....1127 11th St., OALR

SAN DIEGO

BROWN, CHARLES WILLIAM.....Medico-Dental Bldg., ALR
BURTON, FRANK A.....Medico-Dental Bldg., ALR
DAVIS, CHAS. L.....1305 Medico-Dental Bldg., ALR
DENNIS, F. L.....3811 Hawk St., ALR

DURR, S. A.....Medico-Dental Bldg., Op.
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WESSELS, ANDREW BENJAMIN.....Medico-Dental Bldg., ALR

SAN FRANCISCO

BARKAN, ADOLPH.....490 Post St., OALR
BARKAN, HANS.....490 Post St., Op.
BARKAN, OTTO.....490 Post St., Op.
BROWN, H. ALEXANDER.....291 Geary St., Op.
DOLMAN, PERCIVAL.....490 Post St., Op.
GREEN, A. S.....The Green's Eye Hospital, Op.
GREEN, L. D.....The Green's Eye Hospital, Op.
HEBERT, ARTHUR W.....450 Sutter St., OALR
IRVINE, ROBERT STEELE.....490 Post St., ALR
McCOOL, J. L.....450 Sutter St., Op.
SELFRIDGE, G. L.....384 Post St., ALR
SEWALL, E. C.....Stanford Univ. Hospital, ALR
WELTY, C. F.....210 Post St., ALR
ZIEGELMAN, EDWARD FRANK.....450 Sutter St., ALR

SAN JOSE

PUTNAM, F. I.....501 St. Claire Bldg., OALR

SANTA BARBARA

JEAN, G. W.....7 E. Valerio, Op.
JOHNSTON, WM. H.....1421 Chapala St., OALR
LEWIS, J. D.....1534 State St., OALR
MELLINGER, WM. J.....120 E. Sola St., ALR
WELLS, GEORGE S.....8 E. Arrellaga, ALR

SANTA MARIA

BAIRD, CHAS. G.....301 E. Chapel St., OALR

SANTA MONICA

LEVENGOOD, H. WILSON.....327 Wilshire Blvd., OALR

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SMITH, H. A.....315 E. Philadelphia St., OALR

WOODLAND

TILLOTSON, R. S.....Woodland Clinic, ALR

COLORADO

ALAMOSA

HERRIMAN, L. L.....Physicians' Bldg., OALR

BOULDER

FISCHER, V. B.....203 Mercantile Bank Bldg., OALR
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KETTELKAMP, F. O. ..... 301 Ferguson Bldg., ALR
NEEPER, EDW. R. ..... Exchange Nat'l Bank Bldg., Op.

DENVER

ALLEN, J. HENRY ..... Republic Bldg., ALR
BANE, W. C. ..... 1005 Republic Bldg., OALR
BANE, WM. M. ..... 1005 Republic Bldg., OALR
BAUM, H. L. ..... 510 Republic Bldg., ALR
BEYER, T. E. ..... Majestic Bldg., ALR
BLACK, MELVILLE ..... 424 Metropolitan Bldg., Op.
BROWN, MORROW D. ..... 432 Republic Bldg., ALR
CARMODY, T. E. ..... Metropolitan Bldg., ALR
COLLINS, E. W. ..... 508 Majestic Bldg., ALR
COOPER, C. E. ..... 652 Metropolitan Bldg., ALR
CRISP, WM. H. ..... Metropolitan Bldg., Op.
DARROW, C. H. ..... 110 Metropolitan Bldg., ALR
FINNOFF, W. C. ..... Republic Bldg., Op.
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LAFF, H. I. ..... Metropolitan Bldg., ALR
LEVY, ROBERT ..... Metropolitan Bldg., ALR
LEYDA, JAMES H. ..... Metropolitan Bldg., ALR
LOCHARD, L. B. ..... Metropolitan Bldg., ALR
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McGILLICUDDY, OLIVER B. ..... Metropolitan Bldg., ALR
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O'ROURKE, D. H. ..... 217 Imperial Bldg., Op.
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SWERDFEGER, E. B. ..... 1008 Republic Bldg., ALR
WALKER, C. E. ..... 517 Jacobson Bldg., Op.

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RINGLE, CHAS. A. ..... Coronado Block, ALR

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STERLING

PALMER, FRANK E.....301 Main St., OALR

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SMITH, DORLAND.....836 Myrtle Ave., Op.A

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SMITH, E. TERRY.....36 Pearl St., Op.
SNELLING, P. W.....179 Allyn St., ALR
WHALEN, EDWARD J.....750 Main St., ALR

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FLYNN, CHAS. T.....41 Thumbull St., ALR
MACCREADY, PAUL B.....442 Temple St., ALR
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ZINKHAN, A. M.....The Rochambeau, ALR

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BLACK, NELSON MILES.....Huntington Bldg., Op.
CHANDLER, G. E.....Huntington Bldg., ALR
DEBOE, M. P.....1st Nat'l Bank Bldg., OALR
HODSON, B. F.....418 Security Bldg., OALR

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INGERSOLL, J. M.....Aladdin Bldg., ALR
PALMER, BASCOM H.....502 Huntington Bldg., OALR
REPASS, R. E.....337 Lincoln Road, ALR

ORLANDO

INGRAM, L. C.....Orlando Clinic, OALR
SHOEMAKER, SAMUEL A.....30 E. Church St., OALR

PENSACOLA

HEINBERG, CHAS. J.....Blount Bldg., ALR
LISCHKOFF, M. A.....Blount Bldg., OALR

ST. PETERSBURG

HEARD, MARY K.....128 19th Ave. North, Op.

TAMPA

DUKE, R. R.....Citizens' Bank Bldg., ALR
FARRIOR, J. BROWN.....605 Citizens' Bank Bldg., OALR
FORBES, SHERMAN B.....409 Citizens' Bank Bldg., OALR
LOWRY, B. W.....205 Zack St., OALR

WEST PALM BEACH

MILLER, ALICE R.....418 Fern St., OALR

WINTER PARK

OESTERLING, H. E.....642 N. Interlachen Ave., OALR

GEORGIA

ATLANTA

BRAWNER, LEON EDWARD.....157 Forrest Ave., N. E., ALR
CALHOUN, F. PHINIZY.....131 Forrest Ave., Op.
CLAY, G. E.....Medical Arts Bldg., Op.
EQUEN M. S.....Medical Arts Bldg., ALR
FORT, A. G.....Doctors' Bldg., OALR
LOKEY, H. M.....Medical Arts Bldg., OALR
MCDOUGALL, WM. L.....Atlanta Nat'l Bank Bldg., ALR
MARTIN, WM. O. JR.....Medical Arts Bldg., Op.
WARREN, WM. C. JR.....Atlanta Nat'l Bank Bldg., ALR

AUGUSTA

OERTEL, T. E.....S. F. Bldg., OALR

COLUMBUS

BLACKMAR, F. B.....Swift Kyle Bldg., ALR

ROME

SMITH, GEORGE B.....Harbin Hospital, OALR

VALDOSTA

SMITH, J. M.....134½ N. Patterson St., OALR

HAWAII

HONOLULU

MORGAN, JAMES A.....48 Young Bldg., OALR
PINKERTON, FORREST JOY.....46 Young Hotel Bldg., OALR
VAN POOLE, G. MCD.....Young Bldg., OALR

IDAHO

NAMPA

SHAWHAN, GLENN E. ..... Smallwood Bldg., OALR

POCATELLO

CLOTHIER, JOSEPH ..... Kane Bldg., OALR

TWIN FALLS

WETHERBEE, CHARLES ..... Booth Bldg., OALR

ILLINOIS

ALTON

JONES, F. W. ..... 101 Broadway, OALR
MIDDLETON, HARRY E. ..... Times Bldg., OALR

AURORA

DARMER, GEORGE A. ..... 401 Terminal Bldg., OALR

BLOOMINGTON

FOX, RALPH D. ..... Unity Bldg., OALR
GAILEY, WATSON W. ..... Griesheim Bldg., OALR
WATKINS, HAROLD R. ..... Griesheim Bldg., ALR

CHAMPAIGN

FORD, HANBY LEWIS ..... 401 Robeson Bldg., OALR

CHICAGO

ALLEN, THOMAS DYER ..... 122 S. Michigan Blvd., Op.
ANDREWS, A. H. ..... 30 N. Michigan Blvd., OALR
ANDREWS, B. F. ..... 25 E. Washington St., OALR
BALLENGER, H. C. ..... 25 E. Washington St., ALR
BECK, JOSEPH C. ..... 185 N. Wabash Ave., ALR
BEEM, IONE F. ..... 31 N. State St., OALR
BERNHEIMER, BENNO L. ..... 104 S. Michigan Ave., ALR
BOTHMAN, LOUIS ..... 122 S. Michigan Ave., Op.
BRAWLEY, F. E. ..... 30 N. Michigan Blvd., Op.
BREDLAU, ERNEST A. ..... 2753 North Ave., ALR
CARNCROSS, HELEN ..... 58 E. Washington St., Op.
CASTROVIEJO, RAMON ..... 231 W. Washington St., Op.
CAVANAUGH, J. A. ..... Suite 1712-30 N. Michigan Blvd., ALR
CHRISTOPH, CARL H. ..... 30 N. Michigan Blvd., OALR
CLEFF, OSCAR ..... 55 E. Washington St., OALR
CLEMENT, C. C. ..... 30 N. Michigan Blvd., OALR
CROSSLEY, E. R. ..... 30 N. Michigan Blvd., OALR
CUSHMAN, BEULAH ..... 25 E. Washington St., Op.
DAVIDSON, H. P. ..... 1424-30 N. Michigan Ave., Op.
DELPH, JOHN F. ..... 104 S. Michigan Blvd., ALR
DENNIS, GEORGE J. ..... 25 E. Washington St., ALR
DOUGHERTY, CLIFFORD L. ..... 104 S. Michigan Ave., ALR

FAITH, THOMAS.....31 N. State St., OALR
FINDLAY, E. K.....30 N. Michigan Ave., Op.
FISHER, W. A.....231 W. Washington St., OALR
FISKE, GEORGE F.....25 E. Washington St., OALR
FITZGERALD, J. P.....30 N. Michigan Blvd., Op.
FOLK, MAX LYON.....55 E. Washington St., OALR
FOWLER, EARL B.....55 E. Washington St., Op.
FOX, NOAH.....841 E. 63rd St., ALR
FRANK, IRA.....104 S. Michigan Blvd., ALR
FULLER, HARRY B.....2219 E. 70th Place, OALR
GAMBLE, RICHARD COTTER.....30 N. Michigan Ave., Op.
GARRAGHAN, EDW. F.....25 E. Washington St., OALR
GEIGER, ARTHUR HENRY.....554 Fullerton Parkway, ALR
GIFFORD, SANFORD R.....58 E. Washington St., Op.
GLATT, MORRIS A.....3254 Lawrence Ave., ALR
GOLDENBURG, MICHAEL.....104 S. Michigan Ave., Op.
GRADLE, H. S.....58 E. Washington St., Op.
GRIM, U. J.....30 N. Michigan Blvd., OALR
GUTTMAN, MELVIN REESE.....185 N. Wabash Ave., ALR
HALL, ALICE K.....25 E. Washington St., ALR
HASELTINE, BURTON.....122 S. Michigan Ave., OALR
HAWLEY, C. W.....Medical Arts Bldg., OALR
HAYDEN, AUSTIN A.....Suite 2009, 25 E. Washington St., OALR
HOFFMAN, L. G.....30 N. Michigan Blvd., OALR
HOLLENDER, A. R.....30 N. Michigan Blvd., ALR
JORDAN, GEORGE T.....30 N. Michigan Blvd., OALR
KAHN, MYRON E.....104 S. Michigan Ave., ALR
KILLEEN, JOHN J.....104 S. Michigan Blvd., ALR
KRAFT, OSCAR H.....1518 N. Dearborn Parkway, Op.
KRONFELD, PETER C.....950 E. Fifty-ninth St., Op.
KULVIN, MAX M.....1 N. Crawford Ave., ALR
LAMPERT, MAX T.....55 E. Washington St., ALR
LEBENSOHN, JAMES E.....25 E. Washington St., OALR
LEDERER, FRANCIS L.....30 N. Michigan Blvd., ALR
LEECH, VERNON M.....55 E. Washington St., Op.
LESHIN, NORMAN.....180 N. Michigan Ave., ALR
LIFSCHUTZ, JACOB.....310 S. Michigan Blvd., OALR
LIVINGSTON, GEORGE S.....185 N. Wabash Ave., ALR
LORING, J. BROWN.....25 E. Washington St., OALR
LYONS, H. R.....30 N. Michigan Blvd., ALR
MCBEAN, GEORGE M.....58 E. Washington St., OALR
MCGINNIS, EDWIN.....104 S. Michigan Blvd., ALR
MARQUIS, GEORGE P.....30 N. Michigan Blvd., ALR
MAYER, LEO LOEB.....104 S. Michigan Ave., Op.
MEYER, SAMUEL J.....58 E. Washington St., Op.
MILLER, T. E.....122 S. Michigan Ave., ALR
MUNDT, G. HENRY.....25 E. Washington St., OALR
MURRAY, A. N.....4753 Broadway, OALR
NANCE, WILLIS O.....30 N. Michigan Blvd., OALR

NOONAN, W. J. ..... 4753 Broadway, OALR
NORCROSS, E. P. ..... 8 S. Michigan Blvd., ALR
NOVAK, FRANK J. JR. ..... 1712, 30 N. Michigan Blvd., ALR
NUGENT, OSCAR B. ..... 231 W. Washington St., Op.
ORCUTT, D. C. ..... 30 N. Michigan Blvd., Op.
PEARLMAN, S. J. ..... 180 N. Michigan Ave., ALR
POLLOCK, H. L. ..... 2551 N. Clark St., ALR
RUSSELL, R. D. ..... 58 E. Washington St., ALR
SALINGER, SAMUEL ..... 25 E. Washington St., ALR
SCIARRETTA, S. A. ..... 55 E. Washington St., ALR
SHAPIRO, S. L. ..... 185 N. Wabash Ave., ALR
SONNENSCHEIN, R. ..... 180 N. Michigan Blvd., ALR
SPIESMAN, IRWIN GABRIEL ..... 25 E. Washington St., ALR
STRAUSS, J. F. ..... 104 S. Michigan Blvd., ALR
SUKER, GEORGE F. ..... 25 E. Washington St., Op.
TAYLOR, LILLIAN E. ..... 25 E. Washington St., ALR
THEOBALD, JOHN JOSEPH ..... 58 E. Washington St., ALR
THEOBALD, W. H. ..... 307 N. Michigan Ave., ALR
THOMSON, VON COLDITZ, G. ..... 25 E. Washington St., OALR
TIVNEN, RICHARD J. ..... 30 N. Michigan Blvd., OALR
VAN DELLEN, A. L. ..... 6857 S. Halsted St., Op.
VON DER HEYDT, ROBERT ..... 25 E. Washington St., Op.
WAGNER, CARL B. ..... 30 N. State St., OALR
WALKER, HERBERT S. ..... 30 N. Michigan Ave., OALR
WILDER, WM. H. ..... 122 S. Michigan Blvd., Op.
WOLF, SAMUEL ..... 55 E. Washington St., Op.
WOOD, CASEY A. ..... 7 W. Madison St., Op.

CHICAGO HEIGHTS

GORRELL, T. J. H. ..... 16th and Vincennes Ave., OALR

DANVILLE

BALDWIN, H. E. ..... Temple Bldg., ALR
CARMODY, THOMAS J. ..... 408 The Temple Bldg., OALR

DIXON

MCNICHOLS, W. A. ..... Dixon Nat'l Bank Bldg., OALR

EAST ST. LOUIS

HANSON, WM. LUDWIG ..... First Nat'l Bank Bldg., ALR
SPITZE, E. C. ..... Murphy Bldg., Op.

EVANSTON

DODD, OSCAR ..... 636 Church St., Op.
GALLOWAY, T. C. ..... 636 Church St., ALR
GREENWOOD, G. J. ..... 708 Church St., ALR
LAWSON, L. J. ..... 636 Church St., ALR
SWAN, C. J. ..... 636 Church St., OALR

FREEPORT

CLARK, J. SHELDON.....509 State Bank Bldg., OALR

GALESBURG

MATHENY, R. C.....306 E. Main St., OALR

GREAT LAKES

WEBER, H. C.....U. S. Naval Hospital, OALR

HOOPESTON

EAREL, A. M.....Willdon Bldg., OALR

HUBBARD WOODS

LOEB, CLARENCE.....1054 Tower Road, Op.

JACKSONVILLE

ADAMS, A. L.....Professional Bldg., OALR

JOLIET

WOODRUFF, H. W.....500 N. Ottawa St., OALR

KANKAKEE

GEIGER, C. W.....Arcade Bldg., OALR

LA SALLE

WOODS, R. H.....Neustadt Bldg., Op.

MACOMB

DUNTLEY, G. S.....103 S. Side Sq., OALR

MATTOON

VOIGT, C. B.....1702 Broadway, OALR

MOLINE

FLATLEY, T. J.....Reliance Bldg., OALR

OAK PARK

THEOBALD, GEORGIANA DVORAK.....715 Lake St., Op.

PEKIN

GLASFORD, S. T.....Farmers' Bank Bldg., OALR

PEORIA

BLICKENSTAFF, A. J.....Peoria Life Bldg., OALR
BROBST, C. H.....Central National Bank Bldg., OALR
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FLOYD, T. W.....811 Peoria Life Bldg., OALR
THOMAS, C. D.....Central National Bank Bldg., OALR
WILLIAMS, W. C.....Jefferson Bldg., OALR

PONTIAC

MIDDLETON, A. B.....Sterry Blk., OALR

QUINCY

GABRIEL, C. K. ..... W. C. U. Bldg., ALR
MERCER, RAY ..... W. C. U. Bldg., OALR
STEVENSON, WALTER ..... W. C. U. Bldg., OALR

ROCKFORD

FRINGER, W. R. ..... William Brown Bldg., Op.
WORMLEY, H. R. ..... Talcott Bldg., OALR

SPRINGFIELD

DEAL, JOHN FRANCIS ..... First Nat'l Bank Bldg., OALR
HAGLER, E. E. ..... 401 East Capitol Ave., OALR

STERLING

BRODRICK, FRANK W. ..... Central Trust Bldg., OALR

STREATOR

LESTER, HARRY S. ..... 420 Murray Bldg., OALR

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LINGEMAN, B. N. ..... Ben Hur Bldg., ALR

EVANSVILLE

BROSE, L. D. ..... 201 S. East First St., OALR
DYER, WALLACE CURTIS ..... Central Union Bank Bldg., ALR
KNAPP, B. J. ..... Citizens' Bank Bldg., OALR
RAVDIN, B. D. ..... Central Union Bank Bldg., OALR
RAVDIN, M. ..... Central Union Bank Bldg., OALR
STANTON, HARMON LAM. ..... Central Union Bank Bldg., OALR

FORT WAYNE

BULSON, A. E. ..... 406 W. Berry St., OALR
BULSON, EUGENE LORING ..... 406 W. Berry St., OALR
MCBRIDE, W. O. ..... 2902 S. Fairchild, Ave., OALR

HAMMOND

KUHN, HEDWIG S. ..... First Trust Bldg., Op.
KUHN, HUGH A. ..... First Trust Bldg., ALR
LLOYD, A. W. ..... 804 Lloyd Bldg., OALR
SHANKLIN, E. M. ..... 575 Hohman St., OALR

INDIANAPOLIS

ADAMS, D. S. ..... 508 Hume-Mansur Bldg., OALR
ARONSON, SIDNEY S. ..... Hume-Mansur Bldg., ALR
BERRY, D. F. ..... Hume-Mansur Bldg., ALR
CARMACK, J. W. ..... Hume-Mansur Bldg., ALR
CHAPPELL, R. S. ..... 305 Traction Terminal Bldg., ALR
CLARK, CECIL P. ..... Hume-Mansur Bldg., Op.
CLEVENGER, W. F. ..... Hume-Mansur Bldg., ALR

CRAFT, KENNETH L.....Hume-Mansur Bldg., ALR
DEARMIN, ROBERT M.....Hume-Mansur Bldg., ALR
DEWEY, C. H., MAJ., M. C.,
U. S. Army Hospital, Ft. Benjamin Harrison, OALR
ELLIS, BERT ED.....Hume-Mansur Bldg., ALR
HUGHES, W. F.....401 Hume-Mansur Bldg., Op.
KEARBY, D. O.....709 Hume-Mansur Bldg., ALR
LARKIN, BERNARD J.....Hume-Mansur Bldg., Op.
LEASURE, JOHN K.....Hume-Mansur Bldg., ALR
LINGEMAN, EDWARD LAUREL.....406 Pennway Bldg., ALR
LITTELL, J. J.....Hume-Mansur Bldg., ALR
McCASKEY, C. H.....Continental Bank Bldg., ALR
MASTERS, ROBERT J.....23 E. Ohio St., Op.
MOLT, WM. F.....529 Bankers Trust Bldg., ALR
MUELLER, LILLIAN BARBARA.....Continental Bank Bldg., ALR
NEWCOMB, JOHN R.....410 Hume-Mansur Bldg., Op.
OVERMAN, F. V.....Hume-Mansur Bldg., ALR
PFAFFLIN, C. A.....445 Bankers Trust Bldg., OALR
ROW, GEORGE S.....906 Hume-Mansur Bldg., Op.
TOMLIN, W. S.....Hume-Mansur Bldg., ALR
VAN OSDOL, HARRY A.....Chamber of Commerce Bldg., ALR
WHITAKER, J.....Hume-Mansur Bldg., OALR
WRIGHT, J. WM.....Hume-Mansur Bldg., ALR

KOKOMO

ADAMS, CHAS. JOSEPH.....101 W. Sycamore St., OALR

LOGANSPORT

EGAN, BURTON WRIGHT.....408 North St., OALR

MARION

BRAUNLIN, WM. H.....Marion Nat'l Bank Bldg., OALR
MILLER, C. E.....Western Reserve Bldg., OALR

MICHIGAN CITY

KRIEGER, G. M.....701 Washington St., OALR

MUNCIE

MILLER, C. E.....917 E. Main St., OALR

SOUTH BEND

BOONE, J. C.....Sherland Bldg., ALR
BOYD-SNEE, HARRY.....716 J. M. S. Bldg., ALR
CASSADY, J. VERNAL.....654 Associates Bldg., OALR
EDWARDS, SLOCOMB R.....Christman Bldg., OALR
LENT, E. J.....122 N. Lafayette Blvd., OALR
SAVERY, C. E.....206 Sherland Bldg., OALR

TERRE HAUTE

ALLEN, ORRIS T.....422 Rose Dispensary Bldg., OALR
GILLUM, J. R.....221 S. 6th St., OALR

MILLER, WM. H.....Tribune Bldg., ALR
RHEIN, ALFRED E.....Tribune Bldg., ALR
STEWART, WALTER E.....Tribune Bldg., OALR

UNION CITY

RUBY, F. M.....OALR

WARSAW

HOWARD, C. N.....Warsaw Clinic, OALR

IOWA

AMES

DYER, BENJAMIN G.....ALR

BURLINGTON

LA FORCE, E. F.....Iowa State Bank Bldg., OALR
YOUNG, H. B.....204 Jefferson St., OALR

CEDAR RAPIDS

BAILEY, FRED W.....Merchants Nat'l Bank Bldg., OALR
IVINS, H. M.....815 Merchants Nat'l Bank Bldg., OALR

CLINTON

WEIH, E. P.....Wilson Bldg., OALR

COUNCIL BLUFFS

DEAN, F. W.....532 First Ave., OALR
HOWARD, LLOYD G.....Council Bluffs Clinic, OALR
MAIDEN, S. D.....403 Bennett Bldg., OALR

CRESTON

CHILDS, H. A.....Eagle Blk., OALR

DAVENPORT

HANDS, S. GILLOTT.....201 West Second St., ALR
HARKNESS, GORDON F.....Putnam Bldg., OALR
HOFMANN, W. P.....707 Kahl Bldg., OALR
LAMB, HARRY HOWE.....American Bank Bldg., ALR

DES MOINES

JONES, CECIL C.....Equitable Bldg., OALR
LYNCH, ROBERT J.....Equitable Bldg., ALR
PEARSON, WM. W.....417 Bankers Trust Bldg., OALR

DUBUQUE

THOMPSON, HOWARD E.....1100 Main St., OALR

FORT DODGE

CHASE, S. B.....Carver Bldg., OALR

INDEPENDENCE

AGNEW, F. F.....120 Third Ave., S. E., OALR

IOWA CITY

ALBRIGHT, GEORGE C.....102 S. Clinton St., ALR
BOILER, W. F.....Dey Bldg., OALR
LIERLE, DEAN McA.....University Hospital, ALR
MOORE, PAUL McNARY, JR.....University Hospital, ALR
O'BRIEN, C. S.....University Hospital, Op.
RUTHERFORD, C. W.....Univ. Hosp., Dept. Ophth., Op.

KEOKUK

LAPSLEY, ROBERT M.....Masonic Temple, OALR

LE MARS

JOYNT, M. J.....Royal Bldg., ALR

MARSHALLTOWN

COBB, EDWIN.....Masonic Temple Bldg., OALR
FRENCH, R. F.....Masonic Temple Bldg., OALR
SINGLETON, E. M.....111 E. Church St., R
WOLFE, OTIS R.....415 Masonic Temple, OALR

MASON CITY

FALLOWS, HOWARD D.....Park Hospital Clinic, OALR
O'BRIEN, STEVE A.....First Nat'l Bank Bldg., OALR

MUSCATINE

GOAD, R. R.....Laurel Bldg., Op.

NEWTON

ENGLE, H. P.....527 West First Ave., OALR

OTTUMWA

TAYLOR, CHARLES B.....118 E. Second St., ALR

SHENANDOAH

WEAVER, JOHN O.....608½ W. Sheridan Ave., ALR

SIOUX CITY

DECKER, J. C.....Frances Bldg., OALR
FRANCHERE, FRED'K. E.....Toy Bank Bldg., OALR
GITTINS, T. R.....401 Davidson Bldg., OALR
NAFTZGER, J. B.....Davidson Bldg., OALR
REEDER, J. E.....Davidson Bldg., OALR
SALLANDER, F. W.....New Orpheum Bldg., OALR
SECOY, F. L.....301 Davidson Bldg., OALR
TRIPP, LEROY R.....Security Bldg., OALR

WATERLOO

BENDER, H. A.....Black Bldg., ALR
JOYNT, A. J.....Black Bldg., OALR
REULING, F. H.....Black Bldg., OALR
RUSSELL, R. E.....601 Pioneer Bank Bldg., OALR

WAVERLY

ROBINSON, R. E.....205½ E. Bremer Ave., OALR

KANSAS

ATCHISON

BRIBACH, EUGENE J.....Simpson Bldg., OALR

COFFEYVILLE

CHADWICK, IRA B.....Terminal Bldg., Op.

CONCORDIA

ROBERTSON, EDW. N.....132½ W. 6th St., OALR

ELDORADO

SHULTS, E. J. G.....Haines Bldg., OALR

EMPORIA

GRANGER, W. B.....Citizens Nat'l Bank Bldg., OALR
TRIMBLE, CLARENCE S.....405 Citizens Nat'l Bank Bldg., OALR

HUTCHINSON

GREEVER, B. L.....610 Rorabaugh-Wiley Bldg., OALR
SCALES, H. L.....Rorabaugh-Wiley Bldg., OALR
SCHRANT, J. H.....14 West First St., OALR

KANSAS CITY

LIDIKAY, C. J.....903 N. Seventh St., OALR
SPAKE, LAVERNE B.....322 Brotherhood Bldg., ALR

LAWRENCE

POWELL, LYLE S.....813 Mass. St., OALR

LEAVENWORTH

HAAS, FREDERICK J.....Masonic Bldg., OALR

PRATT

JENKINS, MIKE CLEMONT.....223 S. Main St., ALR

TOPEKA

ALKIRE, H. L.....614 Kansas Ave., OALR
ALLEN, GEORGE V.....835 Kansas Ave., OALR
ALLEN, G. HENRY.....Mills Bldg., OALR
DILLON, IRA H.....A. T. & S. F. Ry. Hospital, Op.
REED, WM. W.....Mills Bldg., ALR

WICHITA

BROWNELL, MORTON EMMONS.....101 N. Main St., Op.
CARTER, E. D.....Central Bldg., ALR
CHENEY, JAMES W.....702 Schweiter Bldg., OALR
GILLETT, WILBUR G.....201½ N. Main St., Op.

GSELL, J. F.....911 Beacon Bldg., OALR
MAGGARD, D. I.....715 Orpheum Bldg., OALR
PALMER, EDW. M.....629 Beacon Bldg., OALR
SEYDELL, E. M.....1030 First Nat'l Bank Bldg., ALR
WEAVER, T. W.....201½ N. Main St., Op.

WINFIELD

RALLS, C. T.....First National Bank Bldg., OALR

KENTUCKY

BEREA

COWLEY, ROBERT HENRY.....OALR

COVINGTON

BLEDSOE, R. W.....1005 Madison Ave., OALR

HARLAN

GUNN, MILUS LYDELL.....Smith Bldg., ALR

LEXINGTON

ARMSTRONG, R. M.....Fayette Bank Bldg., OALR
DEWEESE, CLARENCE.....201 West Second St., OALR
MARKS, SAMUEL BLACKBURN.....Security Trust Bldg., ALR
OFFUTT, W. N.....406 Security Trust Bldg., OALR
YATES, EARL C.....190 N. Upper St., ALR

LOUISVILLE

BASS, A. L.....Heyburn Bldg., OALR
DABNEY, S. G.....Starks Bldg., OALR
DEAN, WALTER.....300 Francis Bldg., OALR
DULANEY, OCTAVIUS.....811 Brown Bldg., ALR
HALL, G. C.....705 Brown Bldg., OALR
PFINGST, A. O.....Heyburn Bldg., Op.
SHAFER, J. J.....Starks Bldg., OALR
VAUGHAN, G. E.....711 S. Third St., OALR
WOLFE, CLAUDE T.....Heyburn Bldg., OALR

NEWPORT

THOMASSON, W. J.....942 York St., ALR

PADUCAH

PACE, ERRETT.....City National Bank Bldg., ALR
REYNOLDS, H. G.....City National Bank Bldg., OALR

RICHMOND

DUNN, MURISON.....OALR

LOUISIANA

LAKE CHARLES

MOSS, OLIN W.....Weber Bldg., ALR

NEW ORLEANS

BAHN, CHAS. A.....1707 Pere Marquette Bldg., Op.
BLUM, HENRY N.....1211 Maison Blanche Bldg., Op.
COLLIER, GEO. BENJAMIN.....1317 Whitney Bldg., ALR
FUCHS, VALENTINE HENRY.....Chaille Bldg., ALR
HASPEL, M. DAVID.....Maison Blanche Bldg., OALR
HUME, JOHN R.....Maison Blanche Bldg., ALR
LANDFRIED, C. J.....607 Baronne Bldg., ALR
LEJEUNE, F. E.....632 Maison Blanche Bldg., ALR
SMITH, VICTOR C.....1707 Pere Marquette Bldg., Op.
TAQUINO, GEORGE J.....208 Audubon Bldg., ALR
WAGNER, W. A.....American Bank Bldg., ALR
WEIL, A. I.....3503 Prytania St., ALR

SHREVEPORT

LARUE, C. L.....Highland Clinic, OALR
MOISE, ALLYN B.....Medical Arts Bldg., ALR
SCALES, J. L.....Medical Arts Bldg., OALR

MAINE

AUGUSTA

TURNER, OLIVER W.....Masonic Temple, OALR

LEWISTON

CHAPPERS, WM. H.....190 Bates St., ALR

PORTLAND

BEACH, S. J.....704 Congress St., Op.
CUMMINGS, GEORGE O.....47 Deering St., ALR
GILBERT, FRANK Y.....148 Park St., OALR
GORDON, CHAS. H.....46 Deering St., ALR
HASKELL, ALFRED W.....129 Park St., Op.
HOLT, E. E., JR.....723 Congress St., Op.
O'SULLIVAN, T. J.....10 Pine St., ALR
SPALDING, J. A.....627 Congress St., Op.A

WATERVILLE

HILL, FREDERICK T.....Professional Bldg., ALR
HILL, HOWARD F.....Professional Bldg., Op.

MARYLAND

ANNAPOLIS

ALBRIGHT, LT.-COM. P. M., M. C., U. S. N.....U.S. Naval Hospital, OALR
WELCH, ROBERT S. G.....86 State Circle, OALR

BALTIMORE

BORDLEY, JAMES, JR.....330 N. Charles St., OALR
FRIEDENWALD, HARRY.....1212 Eutaw Pl., Op.A
FRIEDENWALD, J. S.....1212 Eutaw Pl., Op.

GOLDBACK, L. J. ..... 1012 N. Charles St., Op.
KEMLER, JOS. ..... 1908 Eutaw Pl., OALR
LOOPER, EDWARD ANDERSON ..... 104 Madison St., OALR
PETERMAN, H. E. ..... 518 Cathedral St., OALR
TARUN, WILLIAM ..... 104 W. Madison St., OALR

CUMBERLAND

JONES, E. L. ..... First National Bank Bldg., OALR
ROBINSON, H. T. ..... Medical Bldg., OALR
SHARRETT, GEORGE O. ..... 119 Bedford, OALR

MASSACHUSETTS

BEVERLY

ODENEAL, THOMAS HELM ..... 234 Cabot St., OALR

BOSTON

BRYANT, ALICE GERTRUDE ..... 502 Beacon St., ALR
CARVILL, MAUD ..... 82 Commonwealth Ave., Op.
CHENERY, W. E. ..... 377 Commonwealth Ave., ALR
CORBETT, J. J. ..... 520 Beacon St., OALR
DERBY, GEORGE S. ..... 5 Bay State Road, Op.
DUNPHY, ED. B. ..... 520 Commonwealth Ave., Op.
FREEDMAN, L. M. ..... 475 Commonwealth Ave., ALR
GIBBY, HAROLD J. ..... 27 Elm St., ALR
GREENWOOD, ALLEN ..... 82 Commonwealth Ave., Op.
HURLEY, E. D. ..... 419 Boylston St., Op.
INGLIS, HARRY J. ..... 43 Bay State Road, ALR
KLEINERT, MARGARET NOYES ..... 82 Commonwealth Ave., ALR
KNOWLTON, CHARLES DAVISON ..... 520 Commonwealth Ave., ALR
LANCASTER, W. B. ..... 520 Commonwealth Ave., Op.
LEWIS, J. P. ..... 543 Boylston St., ALR
LOWELL, WM. H. ..... 82 Commonwealth Ave., Op.
LURIE, MOSES H. ..... 483 Beacon St., ALR
MOSHER, HARRIS P. ..... 828 Beacon St., ALR
POIRIER, GEORGE H. ..... 60 Bay State Road, ALR
PORTER, CHAS. T. ..... 520 Commonwealth Ave., ALR
QUINCY, J. E. ..... 270 Commonwealth Ave., ALR
RICE, G. B. ..... 99 Bay State Road, ALR
RICHARDS, L. G. ..... 319 Longwood Ave., ALR
ROWLAND, W. D. ..... 220 Clarendon St., Op.
SCHALL, LEROY A. ..... 270 Commonwealth Ave., ALR
SKIRBALL, J. J. ..... 353 Commonwealth Ave., Op.
SMYTH, DUNCAN C. ..... 375 Commonwealth Ave., ALR
THOMPSON, PETER H. ..... 308 Commonwealth Ave., Op.
TINGLEY, LOUISA PAINE ..... 9 Massachusetts Ave., Op.
TOBEY, GEORGE L., JR. ..... 270 Commonwealth Ave., ALR
WALKER, WALDO W. ..... 99 Bay State Road, ALR
WELLS, DAVID W. ..... Hotel Westminster, Op.

CHICOPEE

GALLAGHER, JOHN H. C.....105 Center St., ALR

CLINTON

MONAHAN, J. A.....181 Chestnut St., OALR

FALL RIVER

RYDER, D. R.....151 Rock St., ALR

FRAMINGHAM

TOMB, EVERETT H.....129 Concord St., ALR

GARDNER

BONE, HERMAN D.....19 Pleasant St., OALR

HOLYOKE

BURKE, JOHN R.....207 Elm St., OALR
HUSSEY, E. J.....245 Maple St., OALR

LAWRENCE

CONLON, F. A.....301 Essex St., OALR

LOWELL

WIGGIN, WM. IRVING.....202 Central St., ALR

LYNN

JONES, E. W.....44 Atlanta St., Op.A
O'REILLY, W. F.....44 S. Common St., OALR

NEW BEDFORD

POTTER, L. F.....68 Walnut St., OALR

NORTHAMPTON

COLLINS, J. D.....187 Main St., ALR
DOW, FRANK E.....4 West St., Op.

PALMER

MOORE, G. A.....353 Main St., OALR

PITTSFIELD

THOMPSON, G. H.....74 North St., OALR

SALEM

HENNESSEY, W. W.....333 Essex St., OALR

SPRINGFIELD

BYRNES, H. F.....67 Chestnut St., OALR
CARLETON, RALPH.....10 Temple St., Op.
IRWIN, VINCENT J., JR.....1634 Main St., OALR
IRWIN, V. J., SR.....1634 Main St., OALR

TAUNTON

BAKER, HARRY B.....59 Main St., OALR

WESTFIELD

DECHTER, M. ARCHIBALD.....Professional Bldg., ALR

WORCESTER

BERRY, GORDON.....36 Pleasant St., ALR
CAHILL, JOHN WM.....390 Main St., OALR
CROISSANT, CHAS. A.....390 Main St., ALR
CROSS, A. E.....390 Main St., Op.
ESTABROOK, CHAS. T.....857 Pleasant St., OALR
HOLZER, WM. F.....36 Pleasant St., Op.
MESSIER, A. EUGENE.....390 Main St., OALR
RICE, JOHN E.....390 Main St., OALR

MICHIGAN

ALPENA

NEWTON, WM. B.....201 N. Second Ave., OALR

ANN ARBOR

FURSTENBERG, A. C.....First Nat'l Bank Bldg., ALR
SLOCUM, GEORGE.....First National Bank Bldg., Op.

BATTLE CREEK

COLVER, B. N.....Sanitarium 1, ALR
FRASER, R. H.....Old Merchants Tower, ALR
HAUGHEY, WILFRED H.....303 Post Bldg., OALR
SLEIGHT, R. D.....303 Post Bldg., Op.
WENCKE, CARL G.....Battle Creek Sanitarium, ALR

BAY CITY

BAKER, CHAS. H.....Bay City Bank Bldg., OALR

DETROIT

AMBERG, EMIL.....622 Maccabee Bldg., ALR
ARBUCKLE, DAVID S.....Henry Ford Hospital, ALR
BARNETT, SAUL E.....25 Parsons St., ALR
BEATTIE, ROBERT.....David Whitney Bldg., OALR
BEGLE, HOWELL L.....2730 East Jefferson Ave., Op.
BENTLEY, NEIL I.....1161 David Whitney Bldg., OALR
BERNSTEIN, E. J.....10 Peterboro, OALR
BROWN, AUDREY O.....742 Maccabee Bldg., OALR
CAMPBELL, D. A.....David Whitney Bldg., OALR
CAMPBELL, DON M.....David Whitney Bldg., OALR
CAMPBELL, M. D.....269 Rowena St., OALR
CARTER, JOHN M.....David Whitney Bldg., ALR
CLIFFORD, T. P.....David Whitney Bldg., OALR
COHOE, D. A.....13535 Woodward Ave., ALR

CONNOR, RAY.....1410 Stroh Bldg., OALR
DEFNET, WM. A.....28 Adams Ave. West, ALR
FOWLER, WM.....Maccabee Bldg., OALR
GLOWACKI, BEN FRANCIS.....Maccabee Bldg., OALR
GOUX, RAYMOND SALOT.....545 David Whitney Bldg., OALR
GRANT, LEE E.....David Whitney Bldg., OALR
HALE, ARTHUR STANLEY.....1609 Eaton Tower, OALR
HARRELL, VOSS.....Hofman Bldg., ALR
HEATH, PARKER.....David Whitney Bldg., Op.
HOWELL, DONALD MACLEAN.....1608 Eaton Tower, ALR
HUGHES, RAY W.....1501 David Whitney Bldg., OALR
KEATING, T. F.....1042 Maccabee Bldg., OALR
KREUTZ, GEORGE C.....Henry Ford Hospital, ALR
MCCLELLAND, CARL C.....1857 David Whitney Bldg., OALR
MAIRE, LEWIS E.....710 Kresge Bldg., OALR
MERCER, R. E.....David Whitney Bldg., LR
MINER, S. G.....2641 E. Jefferson Ave., ALR
MORTON, J. B.....90 E. Warren Ave., OALR
MUNSON, F. T.....2201 E. Jefferson Ave., ALR
O'HORA, B. A.....8-266 General Motors Bldg., ALR
PARKER, WALTER R.....David Whitney Bldg., Op.
PEIRCE, HORACE W.....David Whitney Bldg., ALR
POOS, E. E.....Fisher Bldg., OALR
POTTER, WILLIS A.....7450 Grand River Ave., ALR
RENAUD, G. L.....510 Joseph Campan Ave., OALR
RYERSON, F. L.....Fine Arts Bldg., OALR
SANDERSON, H. H.....David Whitney Bldg., OALR
SHURLY, BURT R.....62 Adams Ave. W., ALR
SIMPSON, H. LEE.....1409 David Whitney Bldg., ALR
SISSON, RAYMOND J.....753 David Whitney Bldg., Op.
SUMMERS, WM. S.....1613 David Whitney Bldg., OALR
SUTHERLAND, JACOB MARION.....General Motors Bldg., ALR
WALKER, CHAS. C.....604 Maccabee Bldg., Op.
WENDEL, J. S.....744 David Whitney Bldg., ALR
WHITNEY, E. L.....Henry Ford Hospital, ALR
WILLSON, W. W.....2204 Eaton Tower, OALR
WOODWORTH, WM. P.....1424 Maccabee Bldg., OALR
WILKINSON, A. P.....664 Fisher Bldg., ALR

ESCANABA

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FLINT

HUBBARD, WM. B.....Dryden Bldg., ALR
STEVENSON, WM. W.....Kresge Bldg., ALR

GRAND RAPIDS

DEAN, ALFRED.....308 Medical Arts Bldg., Op.
GRANT, P. T.....Medical Arts Bldg., OALR
HEETDERKS, D. R.....Medical Arts Bldg., ALR

McRae, J. H.....Grand Rapids Clinic, Op.
Robertson, F. D.....Ashton Bldg., OALR
Roller, Louis A.....446 Paris Ave., S. E., Retired
Snapp, Carl F.....Medical Arts Bldg., ALR

GROSSE POINTE VILLAGE

Merkel, Chas. C.....279 Rivard Blvd., ALR

IONIA

Sedgwick, O. W.....OALR

IRON MOUNTAIN

Boyce, G. H.....First Nat'l Bank Bldg., OALR

JACKSON

Hardie, George C.....290 W. Michigan Ave., ALR
McGarvey, Wm. Edward.....Jackson City Bank Bldg., ALR
Winter, George E.....Sun Bldg., OALR

KALAMAZOO

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Grant, Fred E.....Kalamazoo National Bank Bldg., OALR
Wilbur, Edward P.....Bank of Kalamazoo Bldg., OALR

LANSING

Burdick, Austin F.....330 N. Capitol Ave., OALR
Ellis, C. Ward.....215 N. Walnut St., ALR
Wetzel, John O.....404 Capitol Saving and Loan Bldg., Op.

MENOMINEE

Elwood, Calvin R.....Spies Bldg., OALR
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MT. CLEMENS

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ST. JOSEPH

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SAGINAW

Cortopassi, Andre J.....704 Second Nat'l Bank Bldg., ALR
McKinney, A. R.....704 Second Nat'l Bank Bldg., Op.
Rogers, A. S.....105 S. Jefferson Ave., OALR
Watson, R. S.....120 N. Michigan Ave., OALR

TRAVERSE CITY

Holdsworth, Frank.....Peoples Savings Bank Bldg., OALR

MINNESOTA

ALBERT LEA

FOLKEN, FRANK GARRETT.....306 Albert Lea State Bank Bldg., OALR

BRECKENRIDGE

RICE, CLARENCE PRENTICE.....OALR

CROOKSTON

MORLEY, GEORGE A.....Crookston State Bank Bldg., Op.
OPPEGAARD, C. L.....220 S. Broadway, ALR
PARSONS, J. G.....Crookston Clinic, OALR

DULUTH

KNAPP, F. N.....Bradley Bldg., OALR
SHASTID, T. H.....Sellwood Bldg., Op.
TILDERQUIST, D. L.....425 Bradley Bldg., OALR
WINTER, JOHN A.....205 W. Second St., OALR

FERGUS FALLS

KITTELSON, THEODORE.....Iverson and Lee Bldg., OALR

HIBBING

MORSMAN, L. W.....Morsman Clinic, Op.
SINAMARK, ANDREW.....205 Power Bldg., OALR

MINNEAPOLIS

BORGESON, E. J.....Medical Arts Bldg., Op.
BROWN, EDWARD J.....900 Nicollet Ave., OALR
CAMP, WALTER E.....Medical Arts Bldg., OALR
CAMPBELL, ROBERT A.....520 La Salle Bldg., OALR
CLARK, H. S.....Medical Arts Bldg., OALR
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HANSEN, ERLING W.....Medical Arts Bldg., OALR
HOWARD, W. H.....Medical Arts Bldg., OALR
LITCHFIELD, J. T.....Medical Arts Bldg., OALR
LOOMIS, E. A.....Medical Arts Bldg., OALR
MACNIE, J. S.....74 S. 11th St., OALR
MATTHEWS, JUSTUS.....Medical Arts Bldg., LR
MORTON, HOWARD McI.....Insurance Exchange Bldg., Op.
NEWHART, HORACE.....Medical Arts Bldg., ALR
OBERG, C. M.....Syndicate Bldg., OALR
PATTERSON, W. E.....74 S. 11th St., OALR
PHELPS, KENNETH A.....Medical Arts Bldg., OALR
PRATT, FRED J.....Medical Arts Bldg., OALR
PRATT, JOHN A.....Medical Arts Bldg., OALR
REYNOLDS, J. S.....Medical Arts Bldg., OALR
RUCKER, CHAS. W.....Medical Arts Bldg., Op.
SCHWARTZ, VIRGIL JOSEPH.....Medical Arts Bldg., ALR
SMITH, ARTHUR E.....Donaldson Bldg., OALR

STROUT, E. S. ..... 74 S. 11th St., OALR
STROUT, G. E. ..... 1009 Nicollet Ave., OALR
WATSON, JAMES A. ..... 74 S. 11th St., OALR
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WRIGHT, C. DARCY ..... Medical Arts Bldg., OALR

NEW ULM

REINEKE, GEORGE F. ..... Box 137, OALR

ROCHESTER

ANDERSON, CARL M. ..... Mayo Clinic, ALR
BENEDICT, WILLIAM L. ..... Mayo Clinic, Op.
FIGI, F. A. ..... Mayo Clinic, LR
HEMPSTEAD, BERT E. ..... Mayo Clinic, ALR
HILDING, ANDERSON C. ..... Mayo Clinic, ALR
LILLIE, HAROLD I. ..... Mayo Clinic, ALR
LILLIE, W. IVAN ..... Mayo Clinic, Op.
NEW, GORDON B. ..... Mayo Clinic, L
PRANGEN, AVERY DE H. ..... Mayo Clinic, Op.
STARK, WM. B. ..... Mayo Clinic, ALR
WAGENER, HENRY P. ..... Mayo Clinic, Op.

ST. CLOUD

WENNER, W. T. ..... St. Mary's Bldg., Op.

ST. PAUL

BERRISFORD, PAUL D. ..... Lowry Medical Arts Bldg., OALR
BINGER, H. E. ..... Lowry Medical Bldg., OALR
BOECKMANN, EGIL ..... Medical Arts Bldg., OALR
BRAY, E. R. ..... Lowry Medical Arts Bldg., OALR
BROWN, JOHN C. ..... Medical Arts Bldg., OALR
BURCH, FRANK E. ..... Miller Hospital Clinic, Op.
CONNOR, CHAS. E. ..... Miller Clinic, ALR
FOGARTY, CHAS. W. ..... 372 St. Peter St., OALR
FULTON, JOHN F. ..... 833 Lowry Bldg., OALR
GRANT, H. W. ..... Hamm Bldg., Op.
LARSEN, CARL L. ..... 1027 Lowry Medical Bldg., OALR
LEAVENWORTH, R. O. ..... Lowry Medical Bldg., Op.
LEWIS, WILLIAM W. ..... 836 Lowry Bldg., OALR
NELSON, ALLAN LOUIS ..... 830 Lowry Medical Bldg., Op.
ROTHSCHILD, HAROLD J. ..... 239 Lowry Bldg., OALR
SHELLMAN, J. L. ..... Lowry Medical Arts Bldg., OALR
WHEELER, MERRITT W. ..... 1027 Lowry Bldg., OALR
WOLD, K. C. ..... Lowry Medical Arts Bldg., OALR

WINONA

RISSER, EDWARD D. ..... 310 Choate Bldg., OALR
WALKER, GEORGE H. ..... 172 Main St., OALR

WORTHINGTON

STANLEY, C. R. ..... ALR

MISSISSIPPI

GREENVILLE

MONTGOMERY, D. CAMERON.....Leyser Bldg., ALR

GULFPORT

MCWILLIAMS, CHAS. A.....G. & S. I. Bldg., ALR

JACKSON

ADKINS, GEORGE E.....Jackson Infirmary, ALR
HARRIS, WM. ROBIN.....Lamar Life Bldg., ALR

NATCHEZ

GAUDET, LUCIAN S.....405 Franklin St., OALR

OXFORD

GUYTON, B. S.....512 Van Buren, OALR

VICKSBURG

BELL, M. H.....First National Bank Bldg., Op.
EDWARDS, C. J.....First National Bank Bldg., OALR
HOWARD, E. F.....First National Bank Bldg., ALR
JONES, EDLEY H.....First National Bank Bldg., OALR

MISSOURI

CARTHAGE

POST, WINFRED B.....First National Bank Bldg., OALR
POWERS, EVERETT.....Central National Bank Bldg., OALR

COLUMBIA

SNEED, CARL M.....506 W. Broadway, OALR

EXCELSIOR SPRINGS

SHOEMAKER, FERDINAND.....U. S. Veterans' Hospital, OALR

KANSAS CITY

ALLEN, CHAS. E.....712 Waldheim Bldg., OALR
ALTRINGER, A. N.....Professional Bldg., ALR
BEAL, HOMER A.....1002 Argyle Bldg., ALR
BEIL, J. W.....Bryant Bldg., OALR
BELLOWS, GEORGE E.....1500 Professional Bldg., Op.
BLACK, W. BYRON.....Professional Bldg., ALR
BLAKESLEY, T. S.....Professional Bldg., OALR
CURDY, R. J.....1102 Grand Ave., Op.
CURRAN, E. J.....1805 Fed. Res. Bank Bldg., Op.
DAVIS, HARRY B.....Bryant Bldg., OALR
DIXON, OTTO J.....Professional Bldg., ALR
EUBANK, AMBROSE E.....636 Argyle Bldg., OALR
FOSTER, HAL.....1426 Professional Bldg., ALR
GILLILAND, O. S.....Professional Bldg., ALR

HEDRICK, HAROLD B.....910 Rialto Bldg., ALR
HOWARD, J. W.....Rialto Bldg., OALR
KEITH, W. E.....Professional Bldg., OALR
LANING, J. H.....907 Rialto Bldg., ALR
LEMOINE, A. N.....Rialto Bldg., Op.
LICHTENBERG, J. S.....Rialto Bldg., Op.
LORIE, ALVIN J.....Commerce Bldg., ALR
LUX, PAUL.....409 Commerce Bldg., ALR
MCALESTER, ANDREW W., JR.....Rialto Bldg., Op.
MCCARTY, VIRGIL W.....Professional Bldg., OALR
MCKEE, JOSEPH W.....Professional Bldg., Op.
MILLER, HUGH.....Chambers Bldg., OALR
MOTT, JOHN S.....214 Argyle Bldg., OALR
MYERS, JOHN L.....815 Shukert Bldg., OALR
PAINTER, A. M.....Professional Bldg., ALR
ROBERTS, S. E.....Professional Bldg., ALR
SCHUTZ, W. H.....Bryant Bldg., OALR
SHUMATE, D. L.....500 Argyle Bldg., OALR
SIMPSON, M. B.....Professional Bldg., ALR
THOMASON, H. E.....Rialto Bldg., ALR
TEALL, RAYMOND E.....Professional Bldg., ALR
YAZEL, HERMAN E.....733 Rialto Bldg., OALR

ST. JOSEPH

FORGRAVE, L. ROBERT.....204 Logan Bldg., OALR
MINTON, WM. H.....Carby Bldg., OALR
PROUD, WILLARD C.....Phys. and Surg. Bldg., OALR

ST. LOUIS

ALDEN, ARTHUR M.....Frisco Bldg., ALR
ALVIS, BENNETT Y.....900 Carleton Bldg., Op.
ARBUCKLE, MILLARD F.....1011 Beaumont Med. Bldg., ALR
BARDENHEIER, F. G. A.....Frisco Bldg., ALR
BEISBARTH, CARL C.....3720 Washington Ave., Op.
BIRSNER, LOUIS J.....Missouri Bldg., ALR
BLACK, W. D.....Metropolitan Bldg., ALR
BOEMER, L. C.....3720 Washington Blvd., ALR
BROWN, CLYDE O.....3720 Washington Blvd., ALR
BURNS, S. S.....Missouri Theater Bldg., ALR
CALHOUN, JAMES GRAHAM.....Metropolitan Bldg., Op.
CHARLES, JOS. W.....3720 Washington Ave., Op.
CONE, ALFRED J.....Barnes Hospital, 600 S. Kingshighway, ALR
COSTEN, J. B.....Beaumont Medical Bldg., ALR
DEAN, L. W.....Medical School, Washington Univ., ALR
DOBBS, OTTO ROSS.....2435 N. Grand Blvd., ALR
DONNELL, N. R.....Metropolitan Bldg., Op.
DYER, C. P.....408 Humboldt Bldg., Op.
EBER, CARL THEODORE.....1006 Carleton Bldg., Op.
EIMER, CHARLES EUGENE.....316 Lister Bldg., ALR
FERRIS, DAVID P.....1000 Carleton Bldg., ALR

FINEBERG, MAXWELL.....3858 Westminster Place, ALR
FREIMUTH, L. E.....University Club Bldg., ALR
GLICK, HARRY N.....3115 S. Grand Blvd., ALR
GOLDSTEIN, M. A.....3858 Westminster Place, ALR
GREEN, JOHN.....3720 Washington Blvd., Op.
GREENE, M. L.....Missouri Theater Bldg., Op.
GUGGENHEIM, L. K.....Carleton Bldg., ALR
GUNDELACH, C. A.....825 University Club Bldg., ALR
HANSEL, FRENCH KELLER.....Beaumont Medical Bldg., ALR
HARDESTY, J. F.....706 Missouri Theater Bldg., Op.
HARDY, W. F.....Humboldt Bldg., Op.
HOBART, CARL.....Metropolitan Bldg., Op.
HOOSS, C. H. A.....3115 S. Grand Blvd., Op.
HOURN, GEORGE E.....511 University Club Bldg., ALR
HOWARD, HARVEY J.....School of Med., Washington Univ., Op.
JACOBS, MAX W.....Missouri Theater Bldg., Op.
JAMES, WM. M.....640 S. Kingshighway, Op.
JONES, VINCENT L.....3511 Washington Ave., Op.
KELLEY, ISAAC D., JR.....3720 Washington Blvd., ALR
LAMB, HARVEY D.....826 Metropolitan Bldg., Op.
LEAVY, C. A.....Metropolitan Bldg., ALR
LUEDDE, W. H.....Metropolitan Bldg., Op.
LYMAN, HARRY W.....Carleton Bldg., ALR
MCMAHON, BERNARD JOHN.....Missouri Theater Bldg., ALR
MASON, ROY EARL.....Frisco Bldg., Op.
MILLER, H. ED.....728 Missouri Bldg., ALR
MYERS, EUGENE LEE.....Wall Bldg., ALR
NEMOURS, PAUL ROLAND.....3852 Greer Ave., ALR
PAYNE, R. J.....University Club Bldg., ALR
PELZ, MORT D.....Missouri Bldg., ALR
PFINGSTEN, C. F.....6370 Waterman Ave., ALR
POOS, GROVER H.....Univ. Club Bldg., OALR
POST, LAWRENCE T.....Metropolitan Bldg., Op.
POST, M. HAYWARD.....Metropolitan Bldg., Op.
POTTER, ALLEN B.....3725 S. Kingshighway, ALR
PROETZ, A. W.....Beaumont Bldg., ALR
REIM, HUGO.....417 Metropolitan Bldg., Op.
RYAN, L. M.....Wall Bldg., ALR
SAUER, W. E.....Beaumont Medical Bldg., ALR
SCHLUER, E. P.....Metropolitan Bldg., ALR
SCHOLZ, R. P.....Metropolitan Bldg., ALR
SCHWARTZ, FREDERICK O.....Metropolitan Bldg., Op.
SENSENEY, EUGENE T.....316 Lister Bldg., ALR
SHAHAN, W. E.....Metropolitan Bldg., Op.
SHOEMAKER, W. A.....1006 Carleton Bldg., Op.
SIMON, FREDERICK C.....914 Arcade Bldg., ALR
SMIT, WM. M.....719 Missouri Theater Bldg., ALR
TOOKER, C. W.....Carleton Bldg., Op.
WESTLAKE, SAMUEL B.....Beaumont Med. Bldg., ALR

WIENER, MEYER ..... 900 Carleton Bldg., Op.
WOOD, V. VISSCHER ..... Beaumont Medical Bldg., ALR
WOODRUFF, FREDERICK E. ..... Metropolitan Bldg., Op.

SEDALIA

LOVE, JOHN G. ..... Ilgenfritz Bldg., OALR
TITSWORTH, GUY ..... 111 W. 4th St., OALR

SPRINGFIELD

BAILEY, HAROLD ..... 840 Landers Bldg., OALR
COFFELT, THEODORE A. ..... 1240 Benton Ave., OALR
KLINGNER, THOS. O. ..... 318 Landers Bldg., OALR
SMITH, CARLIE SOUTER ..... 214 N. Jefferson Ave., OALR

TRENTON

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MONTANA

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POTTER, PETER ..... Murray Hospital, OALR

GREAT FALLS

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ROUNDUP

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NEBRASKA

COLUMBUS

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KOEBBE, EZRA EDWIN ..... ALR

GRAND ISLAND

RIGG, JAMES P. ..... Grand Island Clinic, ALR

HASTINGS

MARTIN, ROY DAVID ..... Foote Bldg., ALR

KEARNEY

FOX, CHAS. H. ..... 5 West 21st St., OALR

LINCOLN

BLACK, PAUL.....Stuart Bldg., Op.
BROOKS, EARL B.....939 Stuart Bldg., OALR
FURGASON, A. P.....Richards Block, OALR
HOMPES, J. J.....Sharp Bldg., OALR
SANDERSON, DAVID D.....Stuart Bldg., OALR
THOMAS, JAMES W.....Sharp Bldg., OALR
ZEMER, S. G.....950 Stuart Bldg., OALR

NEBRASKA CITY

STONECYPHER, D. D.....107 S. 9th St., ALR

NORFOLK

GADBOIS, ARTHUR E.....421 Norfolk Ave., OALR
NELSON, LLOYD L.....Fleming Bldg., OALR

OMAHA

BUSHMAN, L. B.....631 City Nat'l Bank Bldg., OALR
CALLEAS, W. F.....1620 Medical Arts Bldg., ALR
CASSIDY, W. A.....Medical Arts Bldg., ALR
DAVIS, J. CALVIN.....425 Aquila Court, OALR
FAIRCHILD, NORA M.....Medical Arts Bldg., Op.
HANEY, WM. P.....1500 Medical Arts Bldg., OALR
JUDD, J. H.....Medical Arts Bldg., Op.
KNODE, A. R.....708 Medical Arts Bldg., ALR
KULLY, B. M.....Medical Arts Bldg., ALR
OWEN, F. S.....Medical Arts Bldg., OALR
POTTER, GEORGE B.....Medical Arts Bldg., OALR
POTTS, J. B.....Medical Arts Bldg., ALR
ROMONEK, PHILIP L.....Medical Arts Bldg., OALR
RUBENDALL, C.....Medical Arts Bldg., OALR
STOKES, WM. H.....Medical Arts Bldg., Op.
SWAB, CHARLES M.....Medical Arts Bldg., Op.
UREN, C. T.....537 City National Bank Bldg., ALR
WHERRY, W. P.....1500 Medical Arts Bldg., OALR

NEVADA

RENO

FULLER, JOHN ANDRE.....First Nat'l Bank Bldg., OALR

NEW HAMPSHIRE

NASHUA

KITTREDGE, FRANK E.....Masonic Temple, ALR
NUTTER, C. F.....16 Amherst St., Op.

NEW JERSEY

ASBURY PARK

BAESEMAN, R. WINFIELD.....(1929) Jersey Central Bldg., ALR
FISHER, JAMES A.....Jersey Central Bldg., OALR
UPHAM, HELEN F.....305 Third Ave., OALR

ATLANTIC CITY

CHARLTON, C. COULTER.....124 S. Illinois Ave., ALR
MCGIVERN, C. S.....109 S. Iowa Ave., ALR
OLMSTEAD, WM. DE F.....1920 Pacific Ave., Op.

BLOOMFIELD

MOORE, JOHN D.....6 Washington St., OALR

CAMDEN

KLINE, ORAM R.....414 Cooper St., ALR

EAST ORANGE

BUVINGER, CHARLES W.....50 Washington St., OALR

HACKENSACK

GREENFIELD, WILLIAM JOHN.....50 Anderson St., ALR

JERSEY CITY

PYLE, WALLACE.....15 Exchange Pl., OALR

LONG BRANCH

CAMPBELL, W. K.....96 Third Ave., OALR

NEWARK

CARDWELL, EDGAR PARMELE.....15 Fulton St., ALR
CHATTIN, J. FRANKLIN.....671 Broad St., Op.
EAGLETON, WELLS P.....15 Lombardy St., ALR
HAHN, WM. H.....15 Lombardy St., Op.
HUGHES, L. W.....965 Broad St., OALR
HURFF, J. W.....86 Washington St., OALR
KEIM, WILLIAM F.....25 Roseville Ave., ALR
O'CONNOR, D. F.....671 Broad St., OALR
ORTON, HENRY B.....24 Commerce St., ALR
PEER, L. A.....965 Broad St., ALR
QUINBY, W. O'G.....14 James St., OALR
SHERMAN, ELBERT S.....671 Broad St., Op.
WOOD, EARL LEROY.....192 Roseville Ave., ALR
ZEHNDER, A. CHARLES.....188 Roseville Ave., Op.

ORANGE

EMERSON, LINN.....Metropolitan Bldg., OALR

PLAINFIELD

CHILDERS, ROBERT JEFFERSON.....604 Park Ave., ALR
VAN HORN, A. F.....514 Central Ave., ALR

SUMMIT.

BURRITT, NORMAN W.....30 Beechwood Road, ALR

TRENTON

ADAMS, C. F.....34 W. State St., OALR
CRANE, J. WELLINGTON.....128 Third St., OALR

UNION CITY

SACCO, ANTHONY G.....440 New York Ave., ALR

NEW MEXICO

ALBUQUERQUE

BREHMER, HARRISON L.....First Nat'l Bank Bldg., OALR

FORT BAYARD

GILL, ARTHUR E.....U. S. Veterans' Hospital, OALR

NEW YORK

ALBANY

BEDELL, ARTHUR J.....344 State St., Op.
DOWLING, J. IVIMEY.....116 Washington Ave., OALR
HINMAN, E. E.....146 Washington Ave., ALR
JUDGE, HARRY V.....46 Willett St., Op.
TEBBUTT, H. K.....240 State St., ALR

BATAVIA

WILSON, ROBERT G.....Kresge Bldg., ALR

BINGHAMTON

KINNE, SANFORD HALSEY.....73 Main St., ALR
RANDALL, HIRAM.....81 Main St., ALR
WATSON, H. DE WITT.....151 Front St., OALR

BROOKLYN

AGAN, WM. B.....510 Third St., Op.
BERNSTIEN, P. F.....1571 Eastern Parkway, ALR
BUONAGURO, MICHAEL J.....589 Lorimer St., Op.
CUTINO, R. M.....165 Clinton St., Op.
DECOSTE, S. H.....1179 Dean St., ALR
DE YOANNA, ALFRED A.....111 Pierrepont St., ALR
EBELING, WM. B.....54 Greene Ave., Op.
EVANS, J. N.....23 Schermerhorn St., Op.
FEASTER, HENRY J.....461 77th St., ALR
GOLDING, JOSEPH E.....170 Hancock St., Op.
GREENFIELD, SAMUEL D.....169 New York Ave., ALR
HANCOCK, J. C.....135 Cambridge Pl., Op.
HARGITT, CHARLES A.....17 Schermerhorn St., Op.
HAUPT, WM. C.....1235 Dean St., ALR
JAMESON, P. C.....139 Montague St., Op.
KRIMSKY, JOSEPH.....263 Eastern Parkway, OALR
KRUSKAL, I. D.....863 Prospect Pl., Op.
LLOYD, RALPH I.....14 Eighth Ave., Op.
LOMBARDO, MELCHIORE.....142 Joralemon St., Op.
MENGER, LOUIS CHARLES.....202 Weirfield St., ALR
O'BRIEN, G. R.....307 Sterling Place, ALR
ROGERS, ROBERT M.....680 St. Johns Pl., Op.

THOMASHEFSKY, M.....135 Eastern Parkway, ALR
WEETH, CHAS. R.....37 Remsen St., ALR

BUFFALO

ANDREWS, H. D.....33 Allen St., Op.
BENNETT, ARTHUR G.....150 N. Pearl St., Op.
BEYER, LOUIS J.....449 Delaware Ave., ALR
BLAAUW, E. E.....190 Ashland Ave., Op.
BOZER, HERRMANN EUGENE.....468 Delaware Ave., ALR
BROWN, C. M.....510 Delaware Ave., ALR
COTT, C. C.....104 Dorchester Road, ALR
COWPER, H. W.....543 Franklin St., Op.
EDSON, R. A.....957 Delaware Ave., Op.
FAIRBAIRN, JOHN F.....925 Delaware Ave., ALR
FLAGG, J. D.....158 Anderson Place, Op.A
GLOSSER, HERBERT H.....448 Franklin St., Op.
HEALY, JOHN F.....925 Delaware Ave., ALR
KOENIG, IVAN J.....40 North St., Op.
LE WIN, THURBER.....112 Linwood Ave., Op.
LEWIS, F. PARK.....454 Franklin St., Op.
LUHR, A. F.....40 W. North St., Op.
MCKEE, OTTO S.....83 Bryant St., ALR
MARCH, CLARA A.....465 Ashland Ave., Op.
PHILLIPS, WM. LINTON.....131 Linwood Ave., Op.
RENNER, W. S.....341 Linwood Ave., ALR
WEED, HARRY M.....196 Linwood Ave., Op.
WURTZ, W. J. M.....333 Linwood Ave., ALR

CORTLAND

CUTLER, FRANKLIN E.....R. D. No. 2, ALR
HIGGINS, R. P.....20 Court St., OALR
WHITE, O. E.....28 N. Main St., ALR

DUNKIRK

JACKLE, ARTHUR F.....63 E. 4th St., Op.

FLUSHING

GOODE, EDWARD ALLEN.....143-28 Sanford Ave., ALR

GENEVA

SPENGLER, JOHN ARTHUR.....423 S. Main St., Op.

GLENS FALLS

BUCK, CLAIR A.....Rogers Bldg.
FIELDING, F. G.....70 Ridge St., ALR

JAMAICA

BENDER, M. S.....11-150 St., ALR

JAMESTOWN

REGER, H. S.....Physicians Bldg., OALR

LOCKPORT

PARKER, HARRY L. ..... 88 East Ave., ALR
RINGUEBERG, E. N. S. ..... 13 Main St., Op.

MIDDLETOWN

HICKS, WM. J. ..... 15 King St., ALR

MT. VERNON

KEEFE, WM. JOHN ..... 110 E. Sidney Ave., ALR
THOMSON, J. J. ..... 531 E. Lincoln Ave., ALR

NEW ROCHELLE

BECK, A. I. ..... 421 Huguenot St., OALR
WOODRUFF, WALTER STUART ..... 421 Huguenot St., ALR

NEW YORK CITY

AEBLI, RUDOLF ..... 30 E. 40th St., Op.
ALMOUR, RALPH W. ..... 51 W. 73rd St., ALR
AUERBACH, JULIUS ..... 1016 Fifth Ave., ALR
BALLIN, M. J. ..... 20 E. 76th St., ALR
BASSEN, ED. J. ..... 698 Madison Ave., Op.
BEACH, B. S. ..... 140 W. 86th St., Op.
BERLINER, M. L. ..... 57 W. 57th St., Op.
BENDER, M. S. ..... 30 E. 40th St., ALR
BERENS, CONRAD ..... 35 E. 70th St., Op.
BRUCE, G. M. ..... 180 Fort Washington Ave., Op.
BRUDER, JOSEPH ..... 57 W. 57th St., OALR
BULLWINKEL, H. GRIFFIN ..... 30 E. 76th St., ALR
CARTER, W. W. ..... 2 E. 54th St., ALR
CARY, ED. F. ..... 171 W. 57th St., ALR
COHEN, MARTIN ..... 1 W. 85th St., Op.
COHN, FELIX ..... 47 W. 87th St., ALR
COLBERT, ROBERT MARTIN ..... 57 W. 57th St., ALR
CORYA, HAROLD W. ..... 30 E. 40th St., ALR
COTT, D. N. ..... 25 Central Ave., St. George, Staten Isl., ALR
CUNNING, DANIEL S. ..... 145 E. 54th St., OALR
DAVIDSON, MORRIS ..... 210 W. 70th St., Op.
DAVIS, A. EDWARD ..... 40 E. 61st St., Op.
DAVIS, G. E. ..... 1060 Fifth Ave., OALR
DENISON, WARD C. ..... 20 E. 53rd St., ALR
DEPPING, CHAS. WM. ..... 17 W. 54th St., ALR
DIXON, G. S. ..... 1150 Fifth Ave., R
DLUGASCH, LOUIS ..... 110 E. 36th St., ALR
DOHERTY, W. B. ..... 150 W. 55th St., Op.
DUNNINGTON, J. H. ..... 30 W. 59th St., Op.
ELLIS, Z. H. ..... 58 East 65th St., Op.
FERNLUND, GEORGE BYRON ..... 8555 118th St., Richmond Hill, OALR
FOWLER, ROBERT HENRY ..... 153 E. 62nd St., ALR
FOWLKES, JOHN WINSTON ..... 30 W. 59th St., ALR
FRANKEL, EDWARD, JR. ..... 152 W. 58th St., ALR

GATEWOOD, W. L.....330 Park Ave., ALR
GIORDANO, A. J.....749 E. 183rd St., ALR
GOLDMAN, A. MILTON.....121 E. 60th St., Op.
GOLDMAN, H. G.....57 W. 58th St., ALR
GOLDSTEIN, ISADORE.....1060 Park Ave., Op.
GOTTLIEB, MARK J.....2 W. 87th St., ALR
GRESSER, EDWARD BELLAMY.....39 Fifth Ave., Op.
GRUSHLOW, ISRAEL.....271 Central Park, W., ALR
HALLETT, G. DEWAYNE.....274 W. 86th St., Op.
HANCOCK, J. C.....2 Rector St., Op.
HARDY, LEGRAND H.....30 E. 40th St., Op.
HARRIS, THOMAS JEFFERSON.....104 E. 40th St., ALR
HARTSHORNE, ISAAC.....30 W. 59th St., Op.
HASKIN, W. H.....57 W. 57th St., ALR
HAYS, HAROLD M.....64 E. 58th St., ALR
HETRICK, L. E.....40 E. 61st St., ALR
HORN, WALTER L.....1040 Park Ave., ALR
HURD, L. M.....39 E. 50th St., ALR
HUGHES, WENDELL LOCHEAD.....20 E. 53rd St., Op.
HUNT, WESTLEY MARSHALL.....33 E. 68th St., ALR
HUTCHINSON, A. T.....30 W. 59th St., ALR
IMPERATORI, C. J.....17 E. 38th St., ALR
INGERMAN, S. M.....418 Central Park, W., OALR
JACOBS, S. M.....1018 E. 163rd St., OALR
JARECKY, HERMAN.....168 W. 86th St., OALR
JEROME, WM. Z.....436 Fort Washington Ave., ALR
JOHNSON, THOMAS HOLLAND.....30 W. 59th St., Op.
JONES, MARVIN F.....121 E. 60th St., ALR
JOSEPHSON, EMANUEL M.....993 Park Ave., ALR
JUDD, H. B.....40 E. 61st St., ALR
KELLY, JOSEPH DOMINIC.....114 E. 54th St., OALR
KEY, BEN WITT.....100 W. 59th St., Op.
KING, J. J.....30 E. 40th St., ALR
KIRBY, D. B.....30 W. 59th St., Op.
KNAPP, ARNOLD.....10 E. 54th St., Op.
KNIGHTON, W. S.....40 E. 61st St., Op.
KOPETZKY, S. J.....51 W. 73rd St., ALR
KRUG, ERNEST F.....988 Fifth Ave., Op.
LA VIGNE, A. A.....471 Park Ave., OALR
LEVINE, JOSEPH.....113 E. 61st St., Op.
LEVY, HARRY H.....1016 Fifth Ave., Op.
LUBMAN, MAX.....17 E. 96th St., ALR
MCCOY, JOHN.....730 Fifth Ave., ALR
MCDANNALD, C. E.....100 W. 59th St., Op.
MCDONALD, DENNIS J.....17 E. 38th St., ALR
MCKEOWN, HUGH S.....30 W. 59th St., Op.
MAY, CHAS. H.....698 Madison Ave., Op.
MAYBAUM, J. L.....1079 Park Ave., ALR
MAYER, EMIL.....320 W. 89th St., ALR

MEEK, R. E. ..... 30 E. 40th St., Op.
MEIERHOF, E. L. (Retired) ..... The Westover, 253 W. 72nd St., OALR
MILLER, JOHN ..... 121 E. 60th St., ALR
MILLER, JOSEPH WILLIAM ..... 305 E. 17th St., ALR
MINSKY, HENRY ..... 1 West 86th St., Op.
MITTENDORF, A. D. ..... 339 Park Ave., Op.
MITTENDORF, W. KEITH ..... 115 E. 53rd St., Op.
MORRISON, WM. WALLACE ..... 39 E. 50th St., ALR
MOYLE, EUGENE H. ..... 1088 Park Ave., ALR
MYERSON, M. C. ..... 12 E. 86th St., ALR
NISSELSON, MAX ..... 754 Beck St., ALR
NORTHCOTT, THOMAS A. ..... 17 E. 38th St., Op.
OAST, SAMUEL P., JR. ..... 30 E. 40th St., Op.
PALMER, ARTHUR ..... 667 Madison Ave., ALR
PETERSON, R. A. ..... 150 Fifth Ave., OALR
REESE, ALGERNON B. ..... 73 E. 71st St., Op.
ROSENBAUM, MORRIS ..... 604 E. Fifth Ave., Op.
ROSENBLUTH, MICHAEL ..... 1018 E. 163rd St., ALR
SAMUELS, BERNARD ..... 57 W. 57th St., Op.
SCAL, JOSEPH COLEMAN ..... 44 W. 74th St., ALR
SCHILLER, A. N. ..... 101 Central Park, West, ALR
SCHLIVEK, KAUFMAN ..... 1016 Fifth Ave., Op.
SCHWARTZ, A. A. ..... 941 Park Ave., ALR
SETTEL, NATHAN ..... 250 W. 94th St., ALR
SEWARD, J. A. ..... 121 E. 60th St., ALR
SMITH, JAS. M. ..... 123 E. 53rd St., ALR
SOMBERG, J. S. ..... 470 Park Ave., Op.
STARK, JESSE B. ..... 45 Park Ave., Op.
STURGES, LEIGH F. ..... 121 E. 60th St., ALR
TUSAK, E. A. ..... 115 E. 90th St., Op.
WACHSBERGER, ALFRED ..... 51 W. 73rd St., ALR
WEEKS, WEBB WILLIAM ..... 20 E. 53rd St., Op.
WEXLER, DAVID ..... 55 E. 86th St., Op.
WHEELER, J. M. ..... 30 W. 59th St., Op.
WIENER, ALFRED ..... 550 Park Ave., OALR
WINCOR, HENRY G. ..... 2021 Grand Concourse, Op.
YANKAUER, SIDNEY ..... 121 E. 60th St., ALR

NIAGARA FALLS

PRICE, NORMAN W. ..... 445 Third St., OALR
ROOKER, A. M. ..... 203 Gluck Bldg., ALR

NYACK

TOMS, S. W. S. ..... 120 S. Broadway, OALR

OLEAN

TINDOLPH, L. W. ..... 101 N. Union St., ALR

ONEIDA

CROCKETT, R. L. ..... 302 Main St., Op.

PLATTSBURG

SCHNEIDER, ANTON S.....56 Court St., Op.A

POUGHKEEPSIE

KRIEGER, WM. A.....35 Market St., OALR

ROCHESTER

BARBER, F.....75 S. Fitzhugh St., Op.
BARLOW, ROY A.....Hiram-Sibley Bldg., ALR
CARROLL, GEORGE G.....5 North Goodman St., OALR
CLARK, L. H.....337 Monroe Ave., OALR
GIPNER, J. F.....53 S. Fitzhugh St., Op.
JONES, L. W.....16 N. Goodman St., OALR
KENNEDY, ELDRED WESTON.....Gas and Electric Bldg., Op.
LERNER, MACY LEVI.....332 Park Ave., Op.
MCDOWELL, N. D.....275 Alexander St., OALR
MORRIS, A. G.....277 Alexander St., ALR
NASH, C. STEWART.....26 S. Goodman St., ALR
SHAPERO, I. M.....289 Alexander St., OALR
SNELL, ALBERT C.....53 S. Fitzhugh St., Op.

SYRACUSE

BRITTEN, G. S.....Medical Arts Bldg., ALR
BROWN, MORTIMER G.....Medical Arts Bldg., ALR
BRUST, H. O.....820 S. Crouse Ave., ALR
CAVE, LINUS S.....221 Physicians Bldg., ALR
FOWLER, S. R.....University Block, ALR
GILLETTE, D. F.....State Tower Bldg., Op.
HALL, EDWIN P.....State Tower Bldg., ALR
HOOPLE, GORDON D.....Medical Arts Bldg., ALR
JOY, HAROLD HENRY.....504 State Tower Bldg., Op.
KAUFFMAN, A. B.....Chimes Bldg., ALR
KLINE, H. G.....State Tower Bldg., ALR
MARLOW, SEARLE B.....1004 State Tower Bldg., Op.
MOORE, ROY S.....Medical Arts Bldg., ALR
RUBENSTEIN, A. H.....Medical Arts Bldg., ALR
WEBSTER, F. R.....720 State Tower Bldg., Op.

TROY

SULZMAN, FRANK MALCOLM.....1831 Fifth Ave., OALR

UTICA

BEATTIE, WILLIAM H.....258 Genesee St., OALR
VALONE, FRANK H.....258 Genesee St., ALR

WATERTOWN

ATKINSON, WALTER S.....168 Sterling St., Op.
HENDERSON, L. E.....168 Sterling St., Op.

WHITE PLAINS

MORIARTY, RICHARD W.....Medical Center, OALR

WOODHAVEN

FROTHINGHAM, EDWARD ..... 8644 94th St., OALR

YONKERS

WOLCOTT, CHAS. C. ..... 291 N. Broadway, ALR

NORTH CAROLINA

ASHEVILLE

GREENE, JOSEPH BERRY ..... Haywood Bldg., ALR

CHARLOTTE

MATHESON, J. P. ..... 6 W. 7th St., ALR
PEELER, C. N. ..... 6 W. 7th St., OALR
SLOAN, HENRY L. ..... 6 W. 7th St., Op.
WHISNANT, A. M. ..... 517 Professional Bldg., OALR

GREENSBORO

REAVES, CHAS. R. ..... Reaves Infirmary, 117 W. Sycamore St., OALR
REAVES, W. P. ..... Reaves Infirmary, 117 W. Sycamore St., OALR

KINSTON

PEERY, VANCE PRICE ..... ALR

MOREHEAD CITY

JOHNSON, OTIS H. ..... OALR

NEW BERN

DANIELS, R. L. ..... Box 685, OALR

RALEIGH

HICKS, VONNIE MONROE ..... Professional Bldg., Op.
WRIGHT, J. B. ..... Professional Bldg., ALR

WILMINGTON

MURPHY, J. G. ..... North Carolina Bank Bldg., OALR

WINSTON-SALEM

DAVIS, THOMAS W. ..... 210 O'Hanlon Bldg., OALR

NORTH DAKOTA

BISMARCK

BUCKINGHAM, TRACY W. ..... ALR
CONSTANS, GEORGE MAURICE ..... Quain and Ramstad Clinic, Op.

FARGO

RINDLAUB, ELIZABETH P. ..... de Lendrecie, OALR

MINOT

MCCANNEL, A. D. ..... 33 Central Ave. W., OALR

OHIO

AKRON

BROWN, L. E. ..... Second Nat'l Bank Bldg., OALR
MOORE, T. K. ..... Second Nat'l Bank Bldg., OALR
STEVENSON, D. W. ..... 165 E. Market St., OALR

ALLIANCE

KING, GEORGE L., JR. ..... 537 Market St., Op.
KING, G. L., SR. ..... 537 E. Market St., OALR

BELLEFONTAINE

BUTLER, ROBERT H. ..... 112 W. Columbus Ave., OALR
HARBERT, JOHN P. ..... 135 N. Main St., OALR

CANTON

HOFFMANN, JOHN N. ..... 309 Brant Bldg., ALR
SCHILD, E. H. ..... Brant Bldg., Op.
TOOT, JOSEPH F. ..... 923 First Nat'l Bank Bldg., Op.

CINCINNATI

ABRAHAMSON, IRA A. ..... 605 Traction Bldg., Op.
ASBURY, MARY K. ..... 3549 Holly Lane, Op.
BEYER, A. G. ..... Doctors Bldg., OALR
BROWN, ALBERT LOUIS ..... Doctors Bldg., Op.
BRYANT, BEN L. ..... 17 Garfield Place, ALR
FISCHBACH, VICTOR W. ..... Union Central Bldg., ALR
GOODYEAR, H. M. ..... Doctors Bldg., ALR
HANS, C. L. ..... Provident Bank Bldg., OALR
HARRIS, W. C. ..... Groton Bldg., ALR
HENDICKS, LOUIS J. ..... 210 W. 12th St., Op.
HOFLING, CHARLES A. ..... 707 Race St., Op.
HOWARD, ROBERT E. ..... Union Central Bldg., ALR
IGLAUER, SAMUEL ..... 707 Race St., ALR
JONES, C. C. ..... 656 Doctors Bldg., ALR
KING, CLARENCE ..... Union Central Bldg., Op.
KING, ED. ..... 1002 Carew Tower, ALR
LYLE, DONALD J. ..... 19 W. 7th St., OALR
MITHOEFER, W. ..... Doctors Bldg., ALR
PHINNEY, F. D. ..... 22 W. 7th St., OALR
RAY, VICTOR ..... 19 W. 7th St., Op.
REID, H. W. ..... 19 Garfield Place, Op.
SELTZ, SAMUEL ..... Provident Bank Bldg., ALR
SIEGEL, FRANCIS X. ..... 901 Union Central Life Bldg., OALR
STANBERRY, HENRY ..... 556 Doctors Bldg., Op.
STEWART, T. M. ..... 901 Union Trust Bldg., OALR
STITT, H. L. ..... 656 Doctors Bldg., OALR
STOLL, KARL ..... 19 West 7th St., ALR
TANGEMAN, H. F. ..... 606 Union Central Life Bldg., ALR
THOMPSON, E. H. ..... 8 W. 9th St., OALR
URNER, M. H. ..... 2600 Union Central Bldg., OALR

VAIL, D. T. JR. ..... 24 E. 8th St., Op.
VAIL, H. H. ..... 24 E. 8th St., ALR

CLEVELAND

ABBOTT, W. J. ..... Carnegie Medical Bldg., OALR
BRUNER, ABRAM BRENEMAN ..... 1214 Guardian Bldg., OALR
BRUNER, WM. E. ..... Guardian Bldg., Op.
BURKE, T. A. ..... Rose Bldg., OALR
CHAMBERLAIN, WEBB PARKS ..... 7405 Detroit Ave., Op.
COGAN, J. E. ..... 707 Rose Bldg., OALR
DIXON, FRED WILLIS ..... 1029 Rose Bldg., ALR
FORSYTHE, S. T. ..... Rose Bldg., OALR
KENDALL, M. RAYMOND ..... Hanna Bldg., ALR
LARGE, S. H. ..... Hanna Bldg., ALR
LENKER, JOHN N. ..... 1020 Huron Road, ALR
MCDONALD, C. L. ..... 964 Rose Bldg., OALR
METZ, ROY B. ..... 1214 Guardian Bldg., Op.
METZENBAUM, MYRON ..... Carnegie Medical Bldg., ALR
MITCHELL, HORACE E. ..... 15701 Detroit Ave., ALR
MONSON, S. H. ..... 1322 B. F. Keith Bldg., OALR
MOORE, PAUL G. ..... Medical Arts Bldg., Op.
MOTTO, MICHAEL PAUL ..... Rose Bldg., Op.
MULLIN, WM. V. ..... Cleveland Clinic, Euclid Ave. at 93rd St., ALR
MUSSUN, W. G. ..... Medical Arts Bldg., ALR
NELSON, CHAS. F. ..... 606 Schofield Bldg., OALR
PITKIN, C. E. ..... Carnegie Medical Bldg., ALR
PRENDERGAST, D. A. ..... Medical Arts Bldg., OALR
QUITTNER, S. S. ..... 10515 Carnegie Ave., OALR
RIEGER, W. H. ..... Hanna Bldg., ALR
ROSENBERGER, HARRY CLIFFORD ..... Rose Bldg., ALR
RUEDEMANN, ALBERT DARWIN ..... Cleveland Clinic, Op.
RUST, E. G. ..... Hanna Bldg., Op.
SHACKLETON, W. E. ..... 2323 Prospect Ave., Op.
SHIRAS, HOWARD H. ..... 624 Guardian Bldg., Op.
SHIREY, O. M. ..... Osborn Bldg., ALR
SIMONDS, O. F. ..... Hanna Bldg., Op.
STOTTER, ARTHUR L. ..... 1148 Euclid Ave., OALR
TRIPP, IRA A. ..... 436 Rose Bldg., OALR
TUCKERMAN, W. C. ..... 733 Osborn Bldg., Op.
TUCKERMAN, W. H. ..... 733 Osborn Bldg., OALR
WAUGH, J. M. ..... Cleveland Clinic, Euclid Ave. at 93rd St., OALR
WEIDLEIN, IVAN F. ..... 614 Osborn Bldg., ALR

COLUMBUS

BEATTY, H. G. ..... 681 E. Broad St., ALR
BROWN, JOHN E. ..... 370 E. Town St., OALR
CLARK, I. G. ..... 188 E. State St., OALR
DAVIS, WM. C. ..... 327 E. State St., Op.
FROST, ALBERT DALBEY ..... 681 E. Broad St., Op.
HARRIS, ED. WM. ..... 681 E. Broad St., ALR

HAUER, A. M.....327 E. State St., ALR
HELFRICH, ED. D.....269 E. State St., ALR
MEANS, R. G.....137 East State St., ALR
POSTLE, C. D.....240 E. State St., ALR
PROUT, A. W.....380 E. Town St., OALR
SANOR, DANIEL G., JR.....206 E. State St., ALR
SILBERNAGEL, C. E.....240 E. State St., OALR
STOUTENBOROUGH, WM. A.....21 E. State St., OALR
SULZER, G. A.....327 E. State St., OALR
THOMAS, FRANCIS W.....327 E. State St., OALR
TIMBERMAN, ANDREW.....Beggs Bldg., OALR

DAYTON

BINKLEY, ROY S.....Fidelity Bldg., OALR
BLACKBURN, W. J.....663 Reibold Bldg., OALR
DUTROW, H. V.....1040 Fidelity Medical Bldg., OALR
FARMER, A. G.....Fidelity Medical Bldg., OALR
FOUTS, JOHN D.....Fidelity Medical Bldg., OALR
HARRIS, H. B.....Fidelity Medical Bldg., OALR
KILBOURNE, P. H.....Fidelity Medical Bldg., OALR
MILLETTE, J. W.....Reibold Bldg., OALR

IRONTON

DUNN, O. B.....Fourth and Railroad Sts., OALR

LAKEWOOD

MERICA, F. W.....14805 Detroit Ave., ALR

LIMA

STUEBER, F. G.....209 W. North St., OALR
STUEBER, PAUL J.....209 W. North St., OALR

LORAIN

MONOSMITH, O. B.....O. P. S. Bldg., OALR

MANSFIELD

GARBER, J. M.....14 N. Mulberry St., OALR
WISE, RALPH C.....News Bldg., OALR

MARIETTA

SAUER, W. W.....231 Third St., OALR

MARION

BRICKLEY, DANIEL W.....Uhler-Phillips Bldg., OALR

MASSILLON

SCOTT, MERLE E.....First Nat'l Bank Bldg., OALR

MIDDLETOWN

WILLIAMS, WALTON H.....Castell Bldg., OALR

NEWARK

HATCH, C. B.....3 W. Church St., OALR

SPRINGFIELD

EASTON, J. C.....First Nat'l Bank Bldg., OALR
HARTLEY, F. A.....302 E. High St., OALR
HOGUE, D. W.....34 W. High St., OALR
MINOR, C. L.....First Nat'l Bank Bldg., OALR

STEUBENVILLE

GOURLEY, G. F.....Sinclair Bldg., OALR

TIFFIN

PORTER, E. H.....85 Madison St., OALR

TOLEDO

ALDERDYCE, WM.....513 Madison Ave., OALR
ALTER, F. W.....219 Fifteenth St., OALR
DENMAN, IRA O.....330 Richardson Bldg., OALR
KING, CHAS. R.....Spitzer Bldg., OALR
LUKENS, CHARLES.....316 Michigan St., OALR
LUKENS, JOHN A.....316 Michigan St., ALR
SNYDER, W. H.....2001 Collingwood Ave., OALR
STEINFELD, A. L.....125 Fifteenth St., OALR
WINGER, IRA B.....225 Michigan St., OALR

WARREN

STIMSON, GEORGE W.....Packard Bldg., OALR

XENIA

MADDEN, REED.....Allen Bldg., OALR
SHIELDS, LAWRENCE.....Allen Bldg., ALR

YOUNGSTOWN

BEARD, HAROLD JOLLY.....510 Dollar Bank Bldg., OALR
HARTZELL, S. M.....Dollar Savings and Trust Co. Bldg., OALR

ZANESVILLE

CULBERTSON, L. R.....Masonic Temple, OALR

OKLAHOMA

BARTLESVILLE

KISER, J. D.....Cent. Nat'l Bank Bldg., OALR

ENID

PIPER, ARTHUR S.....610 S. Monroe, OALR

GUTHRIE

BARKER, CHAS. B.....224½ W. Okla. Ave., OALR

McALESTER

DAVIS, J. E.....Kress Bldg., OALR

MUSKOGEE

FULLENWIDER, C. M.....Barnes Bldg., OALR

OKLAHOMA CITY

DIXON, W. E.....503 Medical Arts Bldg., OALR
FERGUSON, E. S.....902 Medical Arts Bldg., OALR
GUTHRIE, A. L.....Osler Bldg., OALR
MACDONALD, J. C.....301 W. 12th St., OALR
McHENRY, D. D.....604 Medical Arts Bldg., OALR
McHENRY, L. C.....117 N. Broadway, ALR

TULSA

BRASWELL, JAMES C.....Medical Arts Bldg., OALR
COOK, W. ALBERT.....Medical Arts Bldg., OALR
HARALSON, CHARLES HERNDON.....Medical Arts Bldg., Op.
ROTH, ALBERT W.....Medical Arts Bldg., OALR
SMITH, RURIC N.....Medical Arts Bldg., ALR

OREGON

BAKER

GORDON, GLENN G.....First Nat'l Bank Bldg., ALR

EUGENE

NORTON, ARTHUR H.....1162 Willamette, Op.

LA GRANDE

BOUVY, L. B.....Foley Bldg., OALR

PORTLAND

AINSLIE, GEORGE.....Selling Bldg., OALR
BOUVY, HARRY M.....385½ Washington, OALR
BRUERE, GUSTAVE E.....Medical-Dental Bldg., OALR
CARRUTH, HOWARD ERNEST.....Medical Arts Bldg., OALR
DAVIS, RALPH F.....Mayer Bldg., OALR
FENTON, RALPH A.....806 Medical Arts Bldg., OALR
HENTON, G. E.....Morgan Bldg., OALR
JOHNSTON, WILSON.....611 Lovejoy St., OALR
KARKEET, ROBERT B.....1203 Selling Bldg., OALR
KIEHLE, F. A.....406 Taylor St., OALR
KISTNER, F. B.....609 Mayer Bldg., OALR
LUCAS, HAROLD R.....Medical-Dental Bldg., OALR
LUPTON, IRVING MARTIN.....Medical Arts Bldg., OALR
PETHERAM, C. C.....Medical Arts Bldg., ALR
TAYLOR, ED M.....Medical Arts Bldg., Op.
WEEKS, JOHN E.....360 Edgecliff Road (Retired), Op.

SALEM

BAUM, WM. W.....Guardian Bldg., ALR
CLEMENT, L. O.....Guardian Bldg., OALR

PENNSYLVANIA

ALLENTOWN

UHLER, S. MANN.....104 N. Eighth St., ALR

ALTOONA

BUZZARD, JOSIAH F.....1110 13th Ave., Op.
GLOVER, S. P.....1118 Twelfth Ave., OALR

BEAVER

CRUMRINE, NORMAN ROSS.....595 Third St., OALR
SCROGGS, J. J.....218 E. Third St., Op.A

BETHLEHEM

CHASE, W. D.....230 E. Broad St., ALR

BRADFORD

ASH, GARRETT G.....1 Main St., OALR

BUTLER

DOANE, L. L.....357 N. Main St., OALR
HAZLETT, L. R.....Savings and Trust Bldg., OALR

CHARLEROI

FERMAN, JOHN W.....Stahlman Bldg., OALR

CHESTER

CROSS, G. H.....525 Welsh St., Op.
ORR, ADRIAN V. B.....525 Welsh St., ALR

CONNELLSVILLE

HARRISON, FRED H.....308 First Nat'l Bank Bldg., OALR

DU BOIS

BEALS, C. WEARNE.....41 N. Brady St., ALR

ERIE

DELANEY, JAS. H.....138 W. 9th St., Op.
DENNIS, EDWARD P.....309 W. 10th St., OALR
FUST, JOHN H. E.....221 W. 9th St., OALR
HEARD, C. F.....813 Sassafras St., OALR
JACKSON, J. D.....232 W. 8th St., OALR
RUSSELL, J. A. MERLE.....813 Sassafras St., OALR
SCHLINDWEIN, G. WM.....138 W. 9th St., OALR
SHREVE, OWEN M.....162 W. 8th St., Op.

FRANKLIN

JOBSON, G. B.....West Park and Liberty St., OALR

GREENSBURG

FAIRING, JOHN W.....206 Tribune Review Bldg., OALR
McKEE, CLAUD W.....Coulter Bldg., OALR

HARRISBURG

DAILEY, GILBERT L.....618 N. 3rd St., OALR
FARNSLER, H. HERSHEY.....1438 Market St., OALR
GRACEY, GEORGE F.....209 State St., OALR

HAZLETON

REICHE, O. C.....Markle Bank Bldg., OALR

JOHNSTOWN

BARKER, O. G. A.....804 Johnstown Trust Bldg., Op.
GRIFFITH, HAROLD M.....804 Johnstown Trust Bldg., Op.
HARRIS, C. M.....604 Johnstown Trust Bldg., OALR
RUSH, C. C.....907 U. S. Nat'l Bank Bldg., OALR

LEBANON

REESE, MONROE D.....123 N. 8th St., OALR

MIDDLETOWN

GEORGE, HENRY W.....19 N. Union St., Op.

NEW KENSINGTON

MCCONNELL, T. E.....Logan Trust Bldg., OALR
STEIM, J. M.....First Nat'l Bank Bldg., Op.

NORRISTOWN

BEIDEMAN, JOSEPH ELLSWORTH.....Montgomery Trust Arcade, Op.
PRICE, JOHN BEADLE.....824 De Kalb St., ALR

PHILADELPHIA

ADLER, FRANCIS HEED.....313 S. 17th St., Op.
ALEXANDER, GEORGE J.....1930 Chestnut St., ALR
AMANO, KAGEYAS W.....502 Woodland Terrace, ALR
ANNON, W. T.....235 S. 15th St., ALR
APPLEMAN, L. F.....308 S. 16th St., Op.
BABBITT, JAMES A.....1912 Spruce St., ALR
BAER, BEN F., JR.....1927 Spruce St., Op.
BALDWIN, KATE W.....1117 Spruce St., ALR
BALENTINE, P. L.....1524 Chestnut St., Op.
BLAIR, MORTIMER, W.....N. E. Cor. 20th and Chestnut St., Op.
BOEHRINGER, W. H.....700 Central Medical Bldg., OALR
BRICKER, SACKS.....3701 N. Broad St., ALR
BROWN, SAMUEL HORTON.....1930 Chestnut St., Op.
BRUMM, SETH A.....Medical Arts Bldg., OALR
BURNS, L. J.....1930 Chestnut St., ALR
BUTLER, MARGARET F.....1831 Chestnut St., ALR
BUTT, MIRIAM M.....2045 Chestnut St., Op.
CARPENTER, C.....1930 Chestnut St., Op.
CLERF, LOUIS HENRY.....128 S. 10th St., ALR
COATES, GEORGE M.....1721 Pine St., ALR

COHEN, HERMAN B. ..... Spruce and 13th Sts., ALR
COHEN, SAMUEL ..... 2009 Pine St., ALR
COWAN, ALFRED ..... 1930 Chestnut St., Op.
CREIGHTON, WM. J. ..... 258 S. 18th St., Op.
DAVIS, J. L. ..... 135 S. 18th St., ALR
DAVIS, T. C. ..... 3128 N. Broad St., ALR
DEICHLER, LYN W. ..... 1930 Chestnut St., OALR
DINTENFASS, HENRY ..... 1305 Spruce St., ALR
EMBERY, FRANK ..... 1532 Overington St., ALR
ERSNER, MATTHEW S. ..... 1915 Spruce St., ALR
FEWELL, A. G. ..... 1924 Pine St., Op.
FRY, W. E. ..... 1912 Spruce St., Op.
GLEASON, E. B. ..... 2112 Pine St., ALR
GODDARD, H. M. ..... 1531 Spruce St., ALR
GOUTERMAN, JOSEPH I. ..... 1930 Chestnut St., Op.
GOWEN, FRANCIS V. ..... 904 E. Chelton Ave., ALR
GRISCOM, J. MILTON ..... 2213 Walnut St., Op.
HAWMAN, ERLE G. ..... 4051 N. Broad St., Op.
HEED, C. R. ..... 1205 Spruce St., Op.
HEWSON, WM. ..... 1721 Pine St., ALR
HOLLOWAY, THOMAS B. ..... 1819 Chestnut St., Op.
HUNTER, R. J. ..... 2011 Chestnut St., ALR
HUSIK, D. N. ..... 1930 Chestnut St., ALR
JACKSON, CHEVALIER ..... 235 S. 15th St., L
KAUFMAN, A. SPENCER ..... 1923 Spruce St., ALR
KEELER, J. C. ..... Medical Arts Bldg., ALR
KRAUSS, FREDERICK ..... 1930 Chestnut St., OALR
LAESSLE, HENRY A. ..... The Lenox, 13th and Spruce Sts., ALR
LAWRENCE, GRANVILLE A. ..... Medical Arts Bldg., Op.
LEWIS, FIELDING O. ..... 259 S. 17th St., ALR
LOFTUS, JOHN EDWARD ..... 1930 Chestnut St., ALR
LUKENS, ROBERT McD. ..... 1923 Spruce St., ALR
LUONGO, R. A. ..... 1713 Pine St., ALR
McANDREWS, LEO F. ..... 1930 Chestnut St., Op.
MACKENZIE, ALICE V. ..... 1724 Spruce St., OALR
MACKENZIE, GEORGE W. ..... 1724 Spruce St., OALR
MANN, BENJAMIN H. ..... N. E. Corner 20th and Chestnut Sts., Op.
MITCHELL, E. K. ..... 710 W. Lehigh Ave., ALR
MOORE, GEORGE H. ..... 1737 Chestnut St., OALR
MOORE, WILLIAM FREDERICK ..... 255 S. 16th St., ALR
OLSHO, S. L. ..... 235 S. 15th St., OALR
O'REILLY, CHARLES A. ..... 521 Medical Arts Bldg., ALR
PERSKY, ABRAM H. ..... 1922 Spruce St., ALR
PETER, L. C. ..... 1930 Chestnut St., Op.
PONTIUS, P. J. ..... 1831 Chestnut St., Op.
RADCLIFFE, McCLUNEY ..... 1930 Chestnut St., Op.
REESE, WARREN S. ..... 230 S. 21st St., Op.
RIDPATH, ROBERT F. ..... N. E. Cor. 18th and Chestnut Sts., LR
ROMMEL, JOHN C. ..... 3701 N. Broad St., Op.

RUDOLPHY, JAY BESSON.....1924 Pine St., Op.
SARTAIN, PAUL J.....2006 Walnut St., Op.
SCARLETT, HUNTER W.....230 S. 21st St., Op.
SCHNEIDEMAN, T. B.....2045 Chestnut St., Op.
SCHWEINITZ, GEORGE E. DE.....1705 Walnut St., Op.
SENER, WALTER J.....Physicians Bldg., Op.
SHANNON, C. E. G.....1930 Chestnut St., Op.
SHEMELEY, WM. G., JR.....1724 Spruce St., OALR
SHUSTER, BENJAMIN HARRISON.....1930 Chestnut St., ALR
SKILLERN, S. R., JR.....1734 Pine St., ALR
SLOANE, HENRY O.....1717 Pine St., Op.
SPAETH, EDMUND B.....1930 Chestnut St., Op.
SPENCER, W. H.....1830 S. Rittenhouse Sq., ALR
STILES, CHAS. M.....1130 Spruce St., Op.
STOUT, PHILIP S.....269 S. 19th St., ALR
TASSMAN, ISAAC S.....Medical Arts Bldg., Op.
TUCKER, GABRIEL.....326 S. 19th St., B
WAGERS, ARTHUR J.....1429 Spruce St., ALR
WEINSTEIN, M. A.....1309 Spruce St., ALR
WHELAN, G. L.....2100 Walnut St., ALR
WILLIAMS, HORACE J.....5908 Greene St., Germantown, ALR
ZENTMAYER, WILLIAM....Suite 803, S. E. Cor. 20th & Chestnut, Op.

PITTSBURGH

BERNATZ, C. F.....Park Bldg., Op.
BLAIR, W. W.....121 University Pl., Op.
CARSON, W. E.....Jenkins Arcade Bldg., Op.
CONWAY, JOHN M.....5151 Jenkins Arcade, ALR
CURRY, GLENDON E.....Westinghouse Bldg., Op.
DAY, KENNETH M.....121 University Pl., ALR
DAY, EWING W.....121 University Pl., OALR
FISCHER, N. ARTHUR.....623 Union Trust Bldg., ALR
GROSS, JULIUS E.....8030 Jenkins Arcade Bldg., ALR
GUERINOT, A. J.....604 Westinghouse Bldg., OALR
HAGEMANN, JOHN A.....Highland Bldg., ALR
HAMPSEY, A. R.....806 May Bldg., ALR
HECKEL, EDWARD B.....Jenkins Arcade Bldg., Op.
JAQUISH, CHARLES J.....904 Westinghouse Bldg., ALR
JENNINGS, CHAS. W.....Highland Bldg., Op.
KNEEDLER, G. C.....Professional Bldg., ALR
KREBS, ADOLPH.....Jenkins Arcade Bldg., Op.
LINN, J. G.....7026 Jenkins Arcade Bldg., Op.
MCCREADY, J. HOMER.....121 University Pl., ALR
MCKEE, GEORGE J.....Westinghouse Bldg., ALR
MACLACHLIN, A. A.....May Bldg., ALR
MARKEL, J. CLYDE.....Westinghouse Bldg., Op.
MARSHALL, WATSON.....121 University Pl., ALR
MEANOR, WM. C.....619 Westinghouse Bldg., Op.
PATTERSON, ELLEN J.....121 University Pl., ALR

PLUMER, JOHN S.....121 University Pl., Op.
SEEGMAN, SIMON.....Medical Bldg., ALR
SHUMAN, G. H.....Park Bldg., Op.
SIMPSON, JOHN REID.....825 Highland Bldg., ALR
SMITH, STANLEY.....121 University Pl., Op.
STIEREN, E.....626 Union Trust Bldg., Op.
STURM, S. A.....7036 Jenkins Arcade Bldg., Op.
TAYLOR, M. C.....May Bldg., Op.
THORPE, H. E.....8075 Jenkins Arcade Bldg., Op.
TODD, GROVER CLEVELAND.....Jenkins Arcade Bldg., ALR
TURNER, H. H.....902 Professional Bldg., Op.
WEISSER, E. A.....806 May Bldg., Op.
YOST, WALTER MILTON.....Westinghouse Bldg., Op.

RADNOR

POSEY, WM. C.....Op.

READING

HIGH, ISAAC B.....326 N. 5th St., ALR
LANDIS, JAMES E.....517 Walnut St., ALR
RHODE, SOLON L.....238 N. 6th St., OALR
STOCKLER, J. A.....332 N. 5th St., OALR

RIDGWAY

MCALLISTER, J. C.....10 S. Mill St., AL

ROCHESTER

YOST, WALTER M.....Peoples Bank Bldg., Op.

SAYRE

WEINBERGER, NELSON S.....Robert Packer Hospital, OALR

SCRANTON

CORSER, J. B.....345 Wyoming Ave., Op.
CURTIN, EUGENE A.....703 Connell Bldg., OALR
SULLIVAN, J. J., JR.....228 Scranton Life Bldg., ALR
WILLIAMS, M. M.....Medical Arts Bldg., Op.

SUNBURY

BECKER, H. M.....49 S. 4th St., OALR

WARREN

BALL, MICHAEL V.....316 Hazel St., OALR

WASHINGTON

MCCULLOUGH, CLARENCE JOSEPH.....Washington Trust Bldg., ALR
MCKENNAN, JAMES W.....514 Washington Trust Bldg., Op.
MCMURRAY, J. B.....Washington Trust Bldg., OALR

WAYNESBURG

STAHLMAN, FRED C.....Commercial Bldg., OALR

WILKES-BARRE

BUCKMAN, E. U.....70 S. Franklin St., OALR
CARR, GEORGE W.....54 S. Franklin St., OALR
KISTLER, O. F.....43 N. Franklin St., Op.AR
McLAUGHLIN, P. A.....71 S. Washington St., OALR
MARSDEN, W. C.....64 N. Franklin St., ALR
SCHAPPERT, N. L.....59 S. Washington St., OALR

WILLIAMSPORT

FLOCK, HERMAN F. W.....416 Pine St., ALR

YORK

FACKLER, CHAS. L.....11 E. Market St., ALR
KLINEDINST, JOHN FRED.....220 S. George St., Op.A

RHODE ISLAND

NEWPORT

JACOBY, D. P. A.....106 Touro St., Op.AL

PROVIDENCE

ADAMS, FRANK M.....122 Waterman St., ALR
BOLOTOW, NATHAN A.....108 Waterman St., ALR
CAPRON, F. P.....118 Angell St., OALR
HARVEY, N. D.....112 Waterman St., OALR
LEECH, J. W.....369 Broad St., OALR
MESSINGER, H. C.....210 Angell St., Op.
MUNCY, WM. M.....162 Angell St., OALR
PORTER, L. B.....199 Thayer St., OALR
SHARP, BENJ. S.....339 Thayer St., OALR
TINGLEY, LOUISA PAINE.....171 Westminster St., Op.

SOUTH CAROLINA

CHARLESTON

KOLLOCK, C. W.....86 Wentworth St., OALR
TOWNSEND, JOHN FERRARS.....85 Wentworth St., OALR

GREENVILLE

CARPENTER, E. W.....E. North St., OALR
MAULDIN, L. O.....Chamber of Commerce Bldg., OALR

SPARTANBURG

GRAY, E. B.....909 Montgomery Bldg., ALR

SOUTH DAKOTA

CHAMBERLAIN

FARNSWORTH, M. A.....Chamberlain Sanitarium and Hospital, OALR

HOT SPRINGS

DEAN, ARTHUR CLARK.....Hot Springs Clinic, ALR

HURON

GROSVENOR, L. N. ..... 366 Dakota Ave. So., OALR

MITCHELL

BOBB, E. V. ..... 101 First Nat'l Bank Bldg., OALR

RAPID CITY

WALSH, JOSEPH MARK ..... OALR

SIOUX FALLS

GREGG, JOHN B. ..... 612 Security Nat'l Bank Bldg., OALR
KELLER, S. A. ..... Sioux Falls Clinic, OALR

WATERTOWN

JOHNSON, A. E. ..... OALR

YANKTON

HOHF, JULIUS ..... Clinic Bldg., OALR

TENNESSEE

CHATTANOOGA

HACKNEY, F. J. ..... 630 Volunteer Bldg., OALR
HOGSHEAD, J. M. ..... 602 Georgia Ave., OALR
LAWWILL, STEWART ..... Medical Arts Bldg., OALR
STEELE, WILLARD ..... Provident Bldg., OALR

DYERSBURG

BREWER, JOHN D. ..... Baird Brewer General Hospital, OALR

JACKSON

DANCY, ALEXANDER B. ..... First National Bank Bldg., OALR

JOHNSON CITY

JONES, U. G. ..... Corner Market and Boone Sts., ALR

KNOXVILLE

KINCAID, J. H. ..... 421 W. Church Ave., OALR
POTTER, W. W. ..... Medical Bldg., OALR
REAVES, ROBERT G. ..... 422 W. Cumberland Ave., OALR

MEMPHIS

ANTHONY, DANIEL HARBERT ..... 1720 Exchange Bldg., Op.
BLASSINGAME, C. D. ..... Physicians and Surgeons Bldg., ALR
BLUE, J. B. ..... Columbian Mutual Tower Bldg., OALR
ELLETT, E. C. ..... Exchange Bldg., Op.
FARRINGTON, P. M. ..... Phys. and Surg. Bldg., ALR
HARRIS, J. HARLEY ..... Medical Arts Bldg., ALR
HUGHES, J. A. ..... Sterick Bldg., OALR
LEVY, LOUIS ..... 1052 Madison Ave., ALR
LEWIS, A. C. ..... Exchange Bldg., OALR
LEWIS, PHILIP MERIWETHER ..... Exchange Bldg., OALR

McKINNEY, R.....899 Madison Ave., ALR
MINOR, H. F.....Bank of Commerce Bldg., OALR
PAYNE, VIRGIL LEROY.....1018 Madison Ave., ALR
QUALLS, HARLEY W.....Exchange Bldg., OALR
RYCHENER, RALPH O.....1720 Exchange Bldg., Op.
SELIGSTEIN, MILTON B.....801 Union and Planters Bank Bldg., OALR
SHEA, J. J.....1018 Madison Ave., ALR
SIMPSON, W. LIKELY.....Phys. and Surg. Bldg., ALR
STANFORD, J. B.....1052 Madison Ave., OALR
STINSON, WM. DAVID.....805 Medical Arts Bldg., ALR
WATKINS, EDWIN DIAL.....1412 Exchange Bldg., OALR

NASHVILLE

BRYAN, JAMES LESLIE.....919 Bennie-Dillon Bldg., OALR
CULLOM, M. M.....1119 Bennie-Dillon Bldg., OALR
EZELL, H. C.....228 Doctors Bldg., OALR
HASTY, F. E.....Medical Arts Bldg., ALR
KENNON, W. G.....Doctors Bldg., OALR
WARNER, ROBERT JAY.....Doctors Bldg., Op.

TEXAS

AMARILLO

CRUME, JOHN JAS.....320 Amarillo Bldg., ALR
DUNCAN, ROBERT AUSTIN.....339 Amarillo Bldg., ALR
STREIT, AUGUST J.....811 Fisk Bldg., ALR

AUSTIN

BOERNER, MORRIS H.....Norwood Bldg., OALR
HILGARTNER, H. L.....209 Norwood Bldg., Op.
KEY, SAM N.....Norwood Bldg., OALR

BEEVILLE

SCHULZE, E. C.....Beasley Bldg., OALR

BROWNWOOD

ANDERSON, W. B.....Medical Arts Hospital, OALR

CORPUS CHRISTI

STROUD, EDWARD F.....Medical Professional Bldg., ALR

DALLAS

CARPENTER, E. R.....3805 Bowser Ave., OALR
CARY, E. H.....Medical Arts Bldg., OALR
DECHERD, H. B.....1515 Medical Arts Bldg., OALR
McLAURIN, J. G.....Medical Arts Bldg., OALR
McREYNOLDS, JOHN O.....907 Mercantile Bank Bldg., OALR
NEWTON, F. H.....907 Mercantile Bank Bldg., OALR
NELSON, LEO A.....4105 Live Oak St., ALR
SEAY, D. E.....907 Mercantile Bank Bldg., OALR
SELLERS, L. M.....Medical Arts Bldg., ALR

EL PASO

IRVIN, EDGAR H.....903 First Nat'l Bank Bldg., OALR
SCHUSTER, FRANK P.....200 N. Mesa Ave., OALR
SCHUSTER, S. A.....First Nat'l Bank Bldg., ALR

FORT WORTH

GOODMAN, THOMAS L.....Medical Arts Bldg., ALR
GOUGH, ROY H.....Medical Arts Bldg., OALR
HOWARD, EDWARD L.....405 Medical Arts Bldg., OALR
JACKSON, ATRAS E.....Medical Arts Bldg., Op.
RICHARDSON, JAMES JOSEPH.....Cook Hosp. and Beall Clinic, ALR
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